ISSN: 3139-5309 (Online)

Nexus Global Research Journal of Medical Sciences

Journal Homepage: https://nexusglobalresearch.com/index.php/ngrjms

Review Article:

Antisperm Antibodies and Sperm Membrane Potential in Diabetes Mellitus: New Insights on Its Role in Male Infertility

[1]Nnodim Johnkennedy, [1]Amah Henry, [2]Nwaokoro Joakin Chidozie, [2]Uche Olive Iwuagwu, [1]Kanu Stella [1]Nnawuihe Chika, [3]Maduike Chukwunonso Victor,

[1] Department of Medical Laboratory Science, Imo State University, Owerri Imo State.
[2]Department of Public of Health, School of Health, Federal University of Technology Owerri Nigeria
[3]Department of Medical Laboratory Science, Madonna University Elele, Rivers State

ABSTRACT:
Diabetes mellitus is a chronic metabolic condition that is increasingly been recognised as a potential factor to decreased male reproductive function. Erectile dysfunction and hypogonadism are well established reproductive complications of diabetes, but an increasing body of evidence suggests that hyperglycaemia, oxidative stress, advanced glycation end products, chronic inflammation, mitochondrial dysfunction and disruption of the blood-testis barrier may directly impair spermatogenesis and sperm function. Among the biological repercussions of these disturbances, the modification of the integrity and the membrane potential of the spermatozoa membrane is of special importance as sperm membrane potential is involved in capacitation, ion homeostasis, motility, calcium signalling and the acrosome reaction. Oxidative stress from diabetes may change membrane lipids, ion channels and mitochondrial function, which could affect the electrical and metabolic properties necessary for optimal sperm function. Simultaneously, the rupture of blood–testis or blood–epididymal barriers may result in the exposure of normally shielded sperm antigens to the immune system and the formation of antisperm antibodies. Antisperm antibodies can attach to sperm surface antigens and interfere with sperm motility, sperm capacitation, cervical mucus penetration, sperm-zona pellucida interaction and fertilisation. But direct evidence for a molecular relationship between diabetes, antisperm antibody production and sperm membrane potential is still lacking. This review describes the biological basis of sperm membrane potential, and the effects of diabetes on sperm membrane potential and mitochondrial function, and the immunological basis of antisperm antibody formation and the possible interactions between metabolic and immunological mechanisms in diabetic male infertility. The research suggests that antisperm antibodies and sperm membrane potential may be complimentary indicators for various but related aspects of diabetic reproductive failure. Future studies should examine simultaneously glycaemic control, antisperm antibodies, sperm plasma-membrane potential, mitochondrial membrane potential, oxidative stress and conventional semen parameters to determine if these biomarkers provide additional information about fertility impairment in men with diabetes.

Keyword: antisperm antibodies, diabetes mellitus, male infertility, sperm membrane potential, mitochondrial dysfunction, oxidative stress, hyperglycaemia, sperm capacitation, blood-testis barrier

Citation: Johnkennedy N. *et al.,* (2026). Antisperm Antibodies and Sperm Membrane Potential in Diabetes Mellitus: New Insights on Its Role in Male Infertility, Nexus *Global Research Journal of Medical Sciences, 2*(5), 155–161.

INTRODUCTION:

Diabetes mellitus (DM) is a severe metabolic illness with persistent perturbations of glucose homeostasis and related abnormalities in lipid metabolism, inflammation, vascular function and cellular redox balance. Its repercussions on the cardiovascular, renal, neurological and ophthalmic systems have been well researched, but its impact on male reproductive health has been less well examined over the years. Clinical and experimental studies have shown that diabetes impairs male reproductive function through endocrine abnormalities, oxidative stress, defective spermatogenesis, testicular structural damage, mitochondrial dysfunction, vascular damage and hypothalamic–pituitary–gonadal axis disturbances [1].

A systematic review of hyperglycaemia-associated reproductive dysfunction identified oxidative stress, advanced glycation end products (AGEs), mitochondrial dysfunction, endoplasmic-reticulum stress and changes in cellular signalling as mechanisms that may be implicated in diabetes-associated male reproductive impairment. In recent reviews, oxidative stress, chronic inflammation, AGEs, mitochondrial dysfunction and

***Corresponding Author:** Nnodim Johnkennedy
Received: 30 Mar 2026 | **Accepted:** 02 May 2026 | **Published:** 15 May 2026
Copyright © 2026 The Author(s): This work is licensed under a Creative Commons Attribution- Non-Commercial-No Derivatives 4.0 (CC BY-NC-ND 4.0) International License

blood–testis barrier disruption have all been mentioned as key pathways of diabetic testicular damage [2].

Spermatozoa are particularly susceptible to metabolic perturbations because they have very limited cytoplasmic antioxidant capacity and their plasma membranes contain significant quantities of polyunsaturated fatty acids. It can lead to lipid peroxidation, altered membrane fluidity and permeability, damage to proteins and DNA, and disruption of mitochondrial energy generation. Such changes can ultimately affect the motility and fertilising capability of sperm.
A further property of the sperm cell that warrants more investigation is the membrane potential. The sperm plasma membrane is an electrically active structure that contains ion channels and transporters that are involved in potassium, sodium, calcium, chloride and proton movement. Capacitation is also associated with changes in membrane potential, intracellular pH and calcium signalling interactions. Membrane hyperpolarisation has been linked to capacitation and acrosomal responsiveness whereas aberrant membrane electrical activity may contribute to impaired sperm function [3].
"Diabetes can also affect male reproductive immunity. The blood–testis barrier protects developing germ cells from immune detection. The blood–epididymal barrier protects mature sperm. Breakdown of these barriers may allow sperm antigens to be exposed to immunological components and may possibly lead to the generation of ASA. Antisperm antibodies have been related with reduced sperm motility and impairment of numerous phases of fertilisation, however the exact role of these antibodies in human infertility is still under debate [4].

The convergence of these pathways prompts the question: may metabolic failure in diabetes concurrently increase sperm membrane and mitochondrial malfunction and foster an immunological milieu permissive to antisperm antibody formation? If so, then ASA and sperm membrane potential may be complimentary indicators of immunologic and cellular components of diabetic male reproductive failure.

Therefore, this review looks at the possible association of diabetes mellitus, antisperm antibodies and sperm membrane potential, with special emphasis on oxidative stress, mitochondrial dysfunction, injury to membrane lipids, ion-channel dysregulation, disruption of the blood–testis barrier and immunological infertility [5].

MALE REPRODUCTIVE DYSFUNCTION AND DIABETES MELLITUS

The effects of diabetes on male reproductive are diverse. Chronic hyperglycaemia may affect the reproductive system by direct metabolic toxicity and secondary complications such as oxidative stress, vascular dysfunction, neuropathy and hormonal disturbances.

Metabolic disorders related to diabetes may impact the hypothalamic-pituitary-gonadal axis. Changes in hypothalamic and pituitary signalling may alter gonadotropin secretion and, subsequently, testicular steroidogenesis and spermatogenesis. Insulin resistance and obesity, which are associated with type 2 diabetes mellitus (T2DM), may further affect the metabolism and reproductive endocrine function of testosterone.

Diabetes at the testicular level has been connected with damage to Sertoli cells, Leydig cells and developing germ cells. Impaired Sertoli-cell function may impair the nutritive and structural support necessary for spermatogenesis, while Leydig-cell failure may lead to decreased testosterone synthesis. Experimental investigations have also indicated that diabetes may damage the seminiferous epithelium and blood–testis barrier.

The effects aren't limited to sperm production. Diabetes can also affect sperm maturation, motility, DNA integrity, mitochondrial function and membrane properties. Increased sperm oxidative stress, DNA fragmentation and impaired mitochondrial transmembrane potential have been identified in human diabetes males and these abnormalities have been related with lower fertilising potential [6].

These data show that diabetic male reproductive dysfunction should not be seen as a simple hormonal or erectile malfunction. It may instead include problems in the endocrine milieu of the testis or in the molecular and electrophysiological properties of individual spermatozoa [7].

OXIDATIVE STRESS, HYPERGLYCEMIA AND SPERM DAMAGE

Oxidative stress is one of the most commonly postulated reasons for the association between diabetes and male reproductive problems. Several mechanisms contribute to increased ROS production in persistent

hyperglycaemia including accelerated mitochondrial electron leakage, polyol pathway activation, protein glycation, altered NADPH metabolism and activation of inflammatory signalling.

Physiological processes of spermatozoa require regulated amounts of ROS. Low amounts of ROS are involved in capacitation, hyperactivation and acrosome response. However, overproduction of ROS can overwhelm the cellular antioxidant defences and lead to oxidative damage [8].

The sperm membrane is particularly sensitive to damage because of its high content of polyunsaturated fatty acids. Lipid peroxidation can alter the fluidity, permeability and protein architecture of membranes. These alterations may interfere with the membrane receptors, ion channels and transporters necessary for proper sperm physiology. Oxidative stress can potentially damage the mitochondrial membranes. Sperm mitochondria are localised in the midpiece and are responsible for ATP production required for flagellar activity; mitochondrial malfunction may limit energy supply and therefore motility. This lipid peroxidation of mitochondrial membranes may enhance electron leakage and ROS production, so generating a self-amplifying cycle of oxidative damage.

This relationship is vital to understanding membrane potential since membrane potential depends upon controlled ionic gradients and functional membrane structures. Damage of ion channels, membrane lipids or mitochondrial respiration could thus affect both electrical states of sperm plasma-membrane and mitochondria [9]

BIOLOGICAL SIGNIFICANCE OF SPERM MEMBRANE POTENTIAL

The sperm membrane potential is the electrical potential difference across the sperm plasma membrane. It is due to differential in ionic concentrations and selective permeability of membrane.

Spermatozoa possess a large number of ion channels and transport systems that control the fluxes of potassium, sodium, calcium, chloride and protons. These systems are involved in the maintenance and modification of membrane potential and are critical for sperm motility, capacitation and fertilization [10].

During capacitation sperm experience a number of biochemical and physiologically changes including changes in membrane lipid structure, intracellular alkalinisation, increased intracellular calcium and changes in membrane potential. Hyperpolarisation of the sperm membrane is well characterised in experimental models and has been shown in human sperm.

Changes in membrane potential are intimately related to calcium signalling. Calcium is required for sperm motility, hyperactivation and the acrosome reaction. CatSper and potassium channels are among the ion channels that regulate sperm electrical and calcium physiology [11].

So, sperm membrane potential should not be seen as a sole electrical measurement. It is an integrated property of cellular signalling and ion-channel activity and membrane permeability. Membrane potential abnormalities may therefore provide information regarding sperm functional competence not obtained by standard semen analysis [12].

Sperm Membrane Failure and Diabetes

There are various biochemical processes via which diabetes could influence the sperm membrane potential. First, oxidative stress may cause lipid peroxidation. As membrane lipids contribute to the structural context in which ion channels work, lipid alteration might change ion permeability and electrical properties of membrane. Second, hyperglycaemia can lead to the production of AGEs. AGEs interact with cellular receptors and proteins and may increase oxidative stress and inflammation. The accumulation of AGE in reproductive organs has been associated with infertility and may contribute to structural and functional defects of sperm [13].

Third, diabetes is characterised by mitochondrial dysfunction. Mitochondria create an electrochemical gradient needed for the generation of ATP. Disruption of mitochondrial membrane potential may lead to decreased ATP synthesis, increased ROS formation and apoptosis.

Crucially, mitochondrial membrane potential and plasma-membrane potential are related but separate concepts. The mitochondrial membrane potential is the electrochemical gradient across the inner mitochondrial membrane. The sperm plasma-membrane potential is the electrical activity across the cell surface. Metabolic stress

may affect both but they must be analysed and interpreted separately.

There is evidence of higher ROS, caspase activation and DNA fragmentation in diabetic males, with significant disruption of sperm mitochondrial transmembrane potential. These results indicate a particularly critical connection between diabetes and bioenergetic impairment in sperm [14].

The direct effect of diabetes on the plasma-membrane potential of human sperm is less clearly defined. This is a significant research gap since sperm plasma membrane potential is highly correlated with capacitation and acrosomal response.

Antisperm Antibodies and Immune-Mediated Infertility in Men

Antisperm antibodies - Immunoglobulins directed against sperm antigens. They can be found in both men and women and can be detected in serum, seminal plasma or directly on the sperm surface.

Germ cell and mature sperm development is normally protected from improper immune identification by a series of anatomical and immunological obstacles. Immune-regulatory mechanisms in the testis help support tolerance, and the blood-testis barrier protects developing germ cells from systemic immune surveillance.

The breakdown of these protective processes may expose sperm antigens to the immune system. Antisperm antibodies have been associated with trauma, infection, surgery, blockage and other forms of reproductive system harm.
The clinical importance of ASA is still a subject of ongoing inquiry. Current reviews suggest that ASA occur in a relatively small proportion of infertile men and that their relationship with infertility is dependent on the antibody concentration, immunoglobulin class, antigen specificity, the location of the antibody and the functional relevance of the sperm antigen being targeted.

ASA may impact sperm in a number of ways. They include sperm agglutination, impaired progressive motility, interference with penetration of cervical mucus, altered sperm-zona pellucida interaction, and interference with other pre-fertilization processes. Important to keep in mind that the presence of ASA does not always mean immunological infertility. The biological relevance relies on which sperm antigens are targeted, and if the antibodies block a function important for fertilization [15].

Diabetes and the potential of producing antispermantibodies

It is scientifically feasible that diabetes may contribute to ASA production but further direct examination is required.
Diabetes is characterised by a state of persistent oxidative stress, inflammation, vascular dysfunction and tissue injury. More recent information indicates that diabetes might also affect the integrity of the blood-testis barrier. Indeed, rupture of the barrier is a well-known setting for exposure of germ-cell antigens to the immune system. Diabetic testicular damage might therefore conceivably provide a context for immune identification of sperm antigens.
Hyperglycaemia may also alter proteins by glycation. Glycated proteins can have different structure and immunological characteristics. However, a direct association of the ASA production with sperm protein alteration in diabetes has not been conclusively demonstrated.

Oxidative damage may also result in changes in sperm membrane proteins, exposing previously hidden or changed epitopes. Theoretically, if these changed antigens are identified as immunologically foreign, an autoimmune reaction might take place.

However, these mechanisms are not yet established as clinical pathways but rather theories. More convincing evidence exists in the current literature that diabetes affects the blood–testis environment and sperm cells than that diabetes directly causes clinically significant ASA production [16].

Therefore, future study should be particularly designed to examine blood–testis barrier integrity, oxidative stress and semen characteristics, while determining if there is a difference in the prevalence of ASAs between healthy controls, men with poorly controlled diabetes and men with well-controlled diabetes.

Potential Interactions Between Antisperm Antibodies and Sperm Membrane Potential

One potentially relevant but insufficiently studied element of immunological infertility is the link between

antibody binding and sperm membrane electrophysiology.

ASA attach to antigens on the sperm surface. Some target antigens are linked to membrane proteins, receptors, ion channels or proteins involved in sperm-oocyte contact. Theoretical alteration of membrane organization or interference with receptor function by antibody interaction.

If the antibodies interact with the membrane proteins that participate in ion transport they may possibly affect the ion flux and membrane potential. Similarly, indirect effects of antibody-mediated agglutination or immobilisation on membrane dynamics and sperm functional competence are also possible.

But this suggested association should not be overemphasised. Although ASA have been reported to affect sperm function in the literature, direct evidence showing that ASA induce aberrant sperm plasma-membrane potential is few.
Another Biomarker: Mitochondrial Membrane Potential

In diabetic male infertility, the mitochondrial membrane potential is of significant interest. Mitochondria are the main source of ROS (reactive oxygen species) during metabolic stress and energy supply for sperm movement. Loss of mitochondrial membrane potential may compromise oxidative phosphorylation and ATP production, while increasing electron leakage and oxidative damage.

In diabetic men has shown impaired sperm mitochondrial membrane potential with higher ROS, caspase-3 activation and DNA fragmentation. These anomalies were connected with lower fertilising potential of the sperm.
Thus, measurement of mitochondrial membrane potential might supplement standard semen analysis. A male may have apparently acceptable sperm quantity and shape with severe mitochondrial dysfunction compromising functional ability.
Potential options include, for example, laboratory procedures such as flow-cytometry with fluorescent potentiometric dyes. However, such assessments should be thoroughly standardised since discrepancies in dyes, sample preparation, sperm selection and analytical limits are likely to affect results [17].

Conventional semen parameters and antisperm antibodies

Semen analysis remains the primary laboratory test of male fertility. Important indicators are sperm concentration, total and progressive motility, morphology and viability.

The association of ASA with semen parameters has not been totally constant. Decreased sperm concentration and motility have been documented in several studies and meta-analyses in ASA-positive males, although the effects on morphology and viability have been less consistent.

This variety likely reflects differences in patient populations, antibody classes, antibody binding locations, laboratory procedures, and criteria employed to establish ASA positive.

Therefore, the routine semen parameters in diabetic men should be evaluated in the light of metabolic and molecular factors. A thorough examination could include: blood sugar, fasting; haemoglobin A1c (HbA1c); serum testosterone; follicular stimulating hormone and luteinizing hormone; seminal concentration; progressive motility; morphology of sperm; viability of sperm; anti-sperm antibodies; plasma-membrane potential of sperm; mitochondrial membrane potential; fragmentation of sperm DNA; key indicators of oxidative stress; and antioxidant properties[18].
Such an integrated technique could establish if membrane and immunological indicators add anything to standard semen analysis.

Inflammation and immune system dysfunction in diabetes

Diabetes is increasingly being seen as a syndrome of persistent low-grade inflammation. Increased levels of inflammatory mediators, changes in immune-cell function and oxidative stress can have effects on several tissues, including the reproductive system.

Persistent inflammation in the testicular milieu can affect Sertoli-cell and Leydig-cell function and modify the blood–testis barrier. Inflammatory signalling may further promote oxidative stress, thus establishing a

feedback loop between metabolic and immunological damage.

The testis generally has a highly specialised immunological milieu that promotes spermatogenesis but restricts damaging immune responses. In theory, disturbance of this equilibrium would increase the risk of immunological detection of germ-cell antigens.

Thus, immunological dysregulation related to diabetes may be relevant in ASA development even if diabetes itself is not a direct source of ASA production.

Clinical and Laboratory Importance

The potential link between diabetes, ASA and sperm membrane potential is of interest for laboratory medicine and reproductive medicine [19].

Second, in diabetic males with infertility, assessment may need to go beyond regular semen analysis, especially when standard parameters cannot account for reproductive problems.

Second, sperm membrane and mitochondrial potential measurements could reveal functional information related to sperm quality. Such tests may disclose metabolic or bioenergetic problems not visible from concentration and morphology alone.
Third, ASA testing may be explored in selected patients with suspected immunological infertility, particularly when there are risk factors such as past testicular trauma, surgery or infection or blockage. The detection should be evaluated in the light of the sperm binding antibody status and clinical symptoms and not as a standalone diagnosis.
Fourth, metabolic regulation continues to be important. Hyperglycaemia, obesity, dyslipidaemia and other components of metabolic syndrome may result in oxidative and reproductive harm. Better metabolic regulation should theoretically lessen several pathways leading to sperm dysfunction, but focused clinical trials are needed to clarify the particular effects on ASA and sperm membrane potential [20].

CONCLUSION

Diabetes mellitus may affect male reproductive function through the complex combination of metabolic, oxidative, inflammatory, endocrine, vascular and cellular pathways. Of them, sperm membrane disruption and mitochondrial damage represent crucial linkages between chronic hyperglycaemia and impaired sperm functional competence. Sperm membrane potential is particularly relevant since membrane electrical activity is involved in capacitation, ion transport, calcium signalling, motility and the acrosome reaction.

Antisperm antibodies are a separate but perhaps related area of male reproductive failure. Disruption of the blood–testis or blood–epididymal barrier may lead to exposure of sperm antigens to immune surveillance and oxidative and inflammatory states associated with diabetes could hypothetically lead to antigen modification and immune dysregulation. However, direct evidence of the causal relationship between diabetes and the clinically relevant development of ASA is still lacking.
Therefore, the only scientifically plausible explanation at now is that diabetes, sperm membrane malfunction and antisperm antibodies may reflect crossing paths rather than a demonstrated single causal sequence. The evidence suggests diabetes-associated oxidative stress, mitochondrial dysfunction, sperm membrane damage and impairment of spermatogenesis. ASA are established contributors to a subgroup of immunological infertility. Further studies are needed to clarify the direct link between these two domains.

Future investigations should look at concomitant glycaemic status, oxidative stress, integrity of blood-testis barrier, antisperm antibodies, sperm plasma-membrane potential, mitochondrial membrane potential and standard semen parameters. These integrated studies may allow to define whether these biomarkers are able to define different biological phenotypes of diabetic male infertility and whether they are of diagnostic, prognostic or therapeutic monitoring relevance.

REFERENCES:

1. Zhang, Y., Tong, X., Jin, H. and Sun, L. (2025) ‘Diabetic testicular dysfunction and spermatogenesis impairment: mechanisms and therapeutic prospects’, *Frontiers in Endocrinology*, 16, 1653975
2. Vazquez-Levin, M.H., Marín-Briggiler, C.I. and Veaute, C. (2014) ‘Antisperm antibodies: invaluable tools toward the identification of sperm proteins involved in fertilization’, *American Journal of Reproductive Immunology*, 72, pp. 206–218.

3. Yang, Y., Yang, L., Han, X., Wu, K., Mei, G., Wu, B. and Cheng, Y. (2024) 'The regulation role of calcium channels in mammalian sperm function: a narrative review with a focus on humans and mice', *PeerJ*, 12..
4. Agbaje, I.M., Rogers, D.A., McVicar, C.M., McClure, N., Atkinson, A.B., Mallidis, C. and Lewis, S.E.M. (2007) 'Insulin dependent diabetes mellitus: implications for male reproductive function', *Human Reproduction*, 22, pp. 1871–1877.
5. Dena, S.M., Adeleye, A.O., Mohlala, K., Langa, B.C. and Opuwari, C.S. (2025) 'The impact of diabetes mellitus-related oxidative stress on male fertility: a review', *Journal of Diabetes*, 17(10).
6. Sundararaj, S., Rajendran, K., & Khanna, A. (2026). Induced Pluripotent Stem Cell-Derived Cardiomyocytes (hiPSC-CMs) in Translational Research: Current Landscape, Clinical Applications, and Future Paradigms. *IOASD Journal of Medical and Pharmaceutical Sciences, 3*(2), 53-55.
7. Mallidis, C., Johnston, D.A., Huleihel, M. (2009) 'Spermatogenic and sperm quality changes in diabetic men', *Reproduction*, 137, pp. 899–907.
8. Luo, Z., Wang, L., Luo, J., Shi, L., Wang, L. and Xie, G. (2025) 'Antidiabetic agents and male fertility: unraveling the impact of glucose-lowering therapies on sperm quality in diabetic males a narrative review', *Translational Andrology and Urology*, 14, pp. 3387–3401.
9. Mallidis, C., Agbaje, I., Rogers, D. (2009) 'Advanced glycation end-products accumulate in the reproductive tract of men with diabetes', *International Journal of Andrology*, 32, pp. 599–606.
10. Aitken, R.J., Gibb, Z., Mitchell, L.A., Lambourne, S.R., Connaughton, H.S. and De Iuliis, G.N. (2012) 'Sperm motility is lost in an oxidative stress-dependent mechanism and can be rescued by catalase supplementation', *Biology of Reproduction*, 87, pp. 1–10.
11. Zhu, J.L., Cai, Y.Q., Long, S.L., Chen, Z. and Mo, Z.C. (2020) 'The role of advanced glycation end products in human infertility', *Life Sciences*, 255, 117830
12. Alasmari, W., Barratt, C.L.R., Publicover, S.J.. (2023) 'The role of sperm membrane potential and ion channels in regulating sperm function', *Frontiers in Cell and Developmental Biology*, 11, 1151346.
13. Mukherjee, A.G. and Gopalakrishnan, A.V. (2024) 'Anti-sperm antibodies as an increasing threat to male fertility: immunological insights, diagnostic and therapeutic strategies', *Reproductive Sciences*, 31, pp. 3303–3322.
14. Ding, G.L., Liu, Y., Liu, M.E., Pan, J.X., Guo, M.X., Sheng, J.Z. and Huang, H.F. (2015) 'Diabetes causes male reproductive dysfunction via multiple mechanisms', *Endocrinology*, 156(4), pp. 1369–1379..
15. La Vignera, S., Condorelli, R.A., Di Mauro, M.. (2015) 'Reproductive function in male patients with diabetes mellitus', *Journal of Clinical Medicine*, 4, pp. 1773–1790.
16. Excel, D. K., Chisom, O. A., Chinyere, E. T. D., Nwadikor, U. U., Nneka, O. I., & Ihuarulam, O. C. (2025). Microbial Analysis of Three-Leaf Yam (Dioscorea dumetorum): Microbiological Quality and Spoilage Dynamics During Storage. *IOASD Journal of Medical and Pharmaceutical Sciences, 2*(4), 161-164.
17. Aitken, R.J. (2013) 'Molecular mechanisms regulating human sperm function', *Molecular Human Reproduction*, 19, pp. 373–384.
18. Amaral, S., Oliveira, P.J. and Ramalho-Santos, J. (2008) 'Diabetes and the impairment of reproductive function: possible role of mitochondria and reactive oxygen species', *Current Diabetes Reviews*, 4, pp. 46–54.
19. Bohring, C. and Krause, W. (2005) 'The role of antisperm antibodies during fertilization and for immunological infertility', *Chemical Immunology and Allergy*, 88, pp. 15–26.
20. Darszon, A., Nishigaki, T., Beltran, C. and Treviño, C.L. (2011) 'Calcium channels in the development, maturation, and function of spermatozoa', *Physiological Reviews*, 91, pp. 1305–1355.