ISBN: 978-93-95847-28-5

Research and Reviews in Animal Science Volume III

Editors:

Dr. Surendra Kadam

Ms. Ruksar

Dr. Pritesh Gugale

Dr. Sachin M. Yeole

BHUMI PUBLISHING, INDIA **FIRST EDITION: APRIL 2024**

Research and Reviews in Animal Science Volume III

(ISBN: 978-93-95847-28-5)

Editors

Dr. Surendra Kadam

Department of Zoology,

Gokhale Education Society's Art, Commerce

and Science College, Jawhar,

Dist. Palghar, Maharashtra

Ms. Ruksar

Department of Zoology,

Chaudhary Charan Singh Haryana

Agricultural University,

Hisar, Haryana

Dr. Pritesh Gugale

Department of Zoology,

Rayat Shikshan Sanstha's

Dada Patil Mahavidyalaya,

Karjat Dist. Ahmednagar, Maharashtra

Dr. Sachin M. Yeole

Department of Zoology,

M S P Mandal's,

Shri Shivaji College,

Parbhani, Maharashtra

Bhumi Publishing

April, 2024

Title: Research and Reviews in Animal Science Volume III

Editors: Dr. Surendra Kadam, Ms. Ruksar, Dr. Pritesh Gugale, Dr. Sachin M. Yeole

First Edition: April, 2024

ISBN: 978-93-95847-28-5

Published by:

Bhumi Publishing

BHUMI PUBLISHING

Nigave Khalasa, Tal – Karveer, Dist – Kolhapur, Maharashtra, INDIA 416 207

E-mail: bhumipublishing@gmail.com

PREFACE

In the ever-evolving tapestry of scientific inquiry, the field of Animal Science stands as a cornerstone, where the intricacies of the animal kingdom are explored, understood, and celebrated. As we embark on this journey through the pages of "Research and Reviews in Animal Science," we are poised to delve into a realm where curiosity meets discovery, where questions find their answers, and where the pulse of innovation beats vibrantly.

This compendium represents not just a collection of scholarly works, but a testament to the collective efforts of passionate researchers, scholars, and practitioners who dedicate their intellect and expertise to unraveling the mysteries that surround the diverse species with which we share our planet. Within these pages, readers will encounter a rich tapestry of studies, analyses, and insights that span the breadth and depth of Animal Science.

From the intricacies of animal behavior to the dynamics of livestock production systems, from the exploration of nutritional needs to the pursuit of sustainable practices, the topics encapsulated within this volume are as diverse as the creatures they seek to understand. Each chapter represents a thread in the fabric of knowledge, woven together to form a comprehensive mosaic of understanding.

As we navigate the terrain of contemporary research and review in Animal Science, it is imperative to acknowledge the tireless efforts of the contributors whose dedication fuels the advancement of our understanding. Their commitment to excellence, coupled with their relentless pursuit of truth, serves as a guiding light illuminating the path toward scientific enlightenment.

Moreover, in an era marked by unprecedented global challenges, the insights contained within these pages hold profound implications for the well-being of both animals and humans alike. Whether addressing issues of animal welfare, public health, or environmental sustainability, the research delineated herein serves as a catalyst for positive change, inspiring action and advocacy in pursuit of a more harmonious coexistence with the natural world.

As we embark on this intellectual odyssey, I extend my deepest gratitude to the authors, editors, and reviewers whose contributions have shaped this volume into a beacon of knowledge and discovery. May the insights contained within these pages spark curiosity, provoke contemplation, and inspire a renewed commitment to the pursuit of truth.

Editors

TABLE OF CONTENT

GENOPROTECTIVE NATURE OF ISOLATED D-PINITOL FROM GLYCINE MAX L MERR. PLANTS AGAINST DOXORUBICIN-INDUCED GENOTOXICITY EVALUATED BY IN VIVO SPERM SHAPE ABNORMALITY ASSAY

Sudha M*[1], Vinciya T[1], Noorul Alam I[2], Aishwarya P J[1] and Harikrishnan N[3]

[1]Department of Pharmacology, Faculty of Pharmacy, Dr. M.G.R. Educational and Research Institute (Deemed to be University), Chennai, Tamil Nadu, India.

[2]Department of Pharmaceutics, College of Pharmacy,
Mother Theresa Post Graduate and Research Institute of Health Sciences, Puducherry.

[3]Department of Pharmaceutical Analysis, Faculty of Pharmacy, Dr. M.G.R. Educational and Research Institute (Deemed to be University), Chennai, Tamil Nadu, India.

*Corresponding author E-mail: kaviyasudha7@gmail.com, sudha.pharm@drmgrdu.ac.in

Abstract:

This research study examined the genoprotective effect of D-Pinitol (D-P) against the genotoxicity caused by Doxorubicin (DOX) in albino mice. Sixty male albino mice were divided into ten groups. For 15 days, 0.9% normal saline was administered to Group I (Control). On 1st, 8th and 15th days, intraperitoneal injection of DOX (5 mg/kg) was administered to Group I (Positive control). For 15 days, D-P 100 mg/kg, 200 mg/kg, 300 mg/kg & 400 mg/kg orally was administered to Group III, IV, V & VI respectively. DOX (5 mg/kg) and D-P 100 mg/kg were administered to Group VII. DOX (5 mg/kg) and D-P 200 mg/kg were administered to Group VIII. DOX (5 mg/kg) and D-P 300 mg/kg were administered to Group IX. DOX (5 mg/kg) and D-P 400 mg/kg were administered to Group X. The role of D-P on DOX-induced genotoxicity was assessed using in vivo Sperm shape abnormalities test. In the animals treated with DOX, a significant ($P<0.001$) rise in sperm shape abnormalities and a significant decrease in sperm count were observed. In comparison to the control group, the groups treated with D-P alone did not change the abnormalities in sperm shape or sperm count. When D-P is combined with DOX, there is a dose-dependent reduction in sperm shape abnormalities and an increase in sperm count compared to DOX alone treated group (Group II). Through its antioxidant, free radical scavenger, and anti-inflammatory properties, D-P was effective in providing protection. Since D-P has exhibited protection against DOX-induced genotoxicity, it can be utilized as a genoprotective agent.

Keywords: D-Pinitol, Doxorubicin, Protective Effect, Genotoxicity, Genoprotective Agent.

Introduction:

Neoplasms appear to have increased in prevalence in economically prosperous nations as a consequence of population, ageing and growing, as well as a rise in the acceptance of cancer-related lifestyle changes like cigarette use, inactivity, and sophisticated eating practices [1]. One of the main causes of death in the globe now is neoplasm [2]. Doxorubicin (DOX), an effective chemotherapeutic medication, is an anthracycline antibiotic that is used to treat a number of human malignancies [3]. By intercalating into DNA, blocking topoisomerase II, and halting the production of DNA and RNA, DOX is able to exert its therapeutic benefits [4]. Apparent toxicity to normal tissues apart from its cytotoxic action to tumor cells restrict DOX's therapeutic use because of inflammatory responses, free radical generation, and oxidative stress [5][6]. As DOX plays a significant role in cancer treatment, it is crucial to lessen its toxicity to normal cells. This can be done by administering simultaneously, such as free radical scavengers, antioxidants and anti-inflammatory agents. Hence, a possible therapeutic approach to DOX-induced toxicity is to reduce oxidative stress, and inflammation [5].

A naturally occurring cyclitol molecule called D-Pinitol (D-P) [7] has been utilized in the traditional practice of Ayurveda medicine for many years [8]. D-P is widely distributed in all regions of soybean plants (*Glycine max* L. Merr., a member of the Leguminosae family), where it is the most abundant soluble carbohydrate [9] [10]. D- P's ability to decrease or inhibit oxidative stress [11] and the inflammatory process [12] is responsible for its most therapeutic actions such as cancer preventive [13], cardioprotective [14], hepatoprotective [15] and renal protective effect [16]. So, the purpose of this investigation was to ascertain if isolated D-P could attenuate the genotoxic effects of DOX in normal tissues.

Material and Methods:

***In vivo* sperm shape abnormality assay:**

Materials required:

Doxorubicin HCL (CIPLA, India), Giemsa stain (Hi-media, India), and Microscope (Olympus Optical Co., Germany).

Animal care and handling:

Swiss albino mice (Sex: Male) weighing 25 to 30 g were housed in cages with a twelve - hour light/dark cycle. The animals were acclimated according to CPCSEA criteria before the study began [17]. According to studies conducted by Hajra *et al.* and Navaaro *et al.*, the DOX and D-P dosages were chosen, respectively [5][18].

Methodology:

Table 1: Treatment Protocol

Groups	Labeled	Treatment
I	Vehicle Control	0.5 ml of 0.9% normal saline
II	Positive Control	Doxorubicin (5 mg/kg), i.p. on 1st, 8th and 15 th days (Positive Control)
III	Test	D-Pinitol (100 mg/kg), p.o. daily
IV		D-Pinitol (200 mg/kg), p.o. daily
V		D-Pinitol (300 mg/kg), p.o. daily
VI		D-Pinitol (400 mg/kg), p.o. daily
VII		Doxorubicin (5 mg/kg), i.p. on 1st, 8th and 15 th days+ D-Pinitol (100 mg/kg), p.o. daily
VIII		Doxorubicin (5 mg/kg), i.p. 1st, 8th and 15 th days + D-Pinitol (200 mg/kg), p.o. daily
IX		Doxorubicin (5 mg/kg), i.p. 1st, 8th and 15 th days + D-Pinitol (300 mg/kg), p.o. daily
X		Doxorubicin (5 mg/kg), i.p. 1st, 8th and 15 th days + D-Pinitol (400 mg/kg), p.o. daily

According to the treatment protocol (Table 1), animals received D-P for 15 days and DOX for 3 days (on first day, eighth day, and fifteenth day) [19]. D-P treated 30 minutes prior to the DOX administration. After twenty-four hours of the last treatment, the animals were sacrificed by exposing them to carbon dioxide inhalation. The epididymis was extracted through laparotomy, and the sperm suspension was made by chopping up the epididymis in 1 ml of normal saline. After staining with 1 percent Giemsa for 30 minutes, the solution was filtered through 80 mm nylon mesh to prepare smears to assess sperm shape abnormalities. A microscope was used to count the morphological abnormalities in sperm shape at 100 X magnification. One thousand sperms were examined for morphological damage in each animal, and the results were represented as a percentage of total abnormalities. Neubauer's hemocytometer was used to count the sperm in the epididymis. The results were expressed as the number of sperms per milligram of epididymis weight [20].

Statistical analysis:

One-way ANOVA for this research was performed statistically using GraphPad Prism software version 8.01. Statistics were evaluated to be significant at P values under 0.05 ($P<0.05$).

Results and Discussion:

Table 2: Number of different types of sperm abnormalities that occurred in treated groups of mice

Nature of Sperms	Group I (Control- 0.9% Normal saline)	Dose in mg/kg								
		Group II (DOX 5)	Group III (D-P 100)	Group IV (D-P 200)	Group V (D-P 300)	Group VI (D-P 400)	Group VII (DOX 5+D-P 100)	Group VIII (DOX 5+D-P 200)	Group IX (DOX 5+D-P 300)	Group X (DOX 5+D-P 400)
Normal	750± 13.66	429± 19.14	760± 10.33	767± 18.32	758± 14.19	745± 13.84	481± 16.56	580± 24.18	617± 15.67	679± 16.71
Amorphous head	27± 2.78	30± 2.898	35± 2.098	22± 1.265	31± 1.549	37± 3.55	23± 2.62	33± 2.295	25± 3.386	39± 2.733
Headless Sperm	95± 5.209	160± 5.663	85± 2.394	77± 3.337	98± 3.204	69± 2.543	158± 2.781	120± 4.803	110± 4.524	98± 3.044
Tailless Sperm	11± 1.033	60± 3.941	19± 1.528	23± 1.183	31± 1.751	16± 1.211	37± 1.77	31± 1.528	30± 3.493	47± 4.597
Bent at cephalo -caudal region	4± 0.816	9± 0.632	1± 0	0	5± 1.342	0	1± 0	5± 1.238	6± 1.633	2± 0.365
Bent tail	110± 2.978	240± 6.557	99± 2.921	108± 3.256	74± 4.107	127± 3.367	290± 5.145	231± 6.218	211± 6.126	135± 3.483
Others	3± 0.73	72± 3.215	1± 0	3± 6.831	3± 8.944	6± 9.309	10± 1.183	0	1±0	0

Mean ± SEM, n=6

Table 3: Effect of DOX and D-P on Sperm abnormalities in mice

Criterion	Group I (Control-0.9% Normal saline)	Dose in mg/kg								
		Group II (DOX 5)	Group III (D-P 100)	Group IV (D-P 200)	Group V (D-P 300)	Group VI (D-P 400)	Group VII (DOX 5+ D-P 100)	Group VIII (DOX 5+ D-P 200)	Group IX (DOX 5+ D-P 300)	Group X (DOX 5+ D-P 400)
% Sperm abnormalities	25± 0.877	57.1± 1.194 a*	24± 0.492 aNS	23.3± 0.398 aNS	24.2± 0.46 aNS	25.5± 0.432 aNS	51.9± 1.088 a*b#	42± 0.996 a*b*	38.3± 0.789 a*b*	32.1± 0.695 a*b*
Sperm Count (×10^6/mg of epididymis)	8.75± 0.363	4.633± 0.326 a*	8.917± 0.162 aNS	8.717± 0.183 aNS	8.883± 0.18 aNS	8.883± 0.101 aNS	4.933± 0.249 a*bNS	5.833± 0.223 a*b@	6.533± 0.243 a*b#	7.85± 0.12 aNSb*

Mean ± SEM, n=6, where a - Group II, III, IV, V, VI, VII, VIII, IX, X compared with Group I. b - Group VII, VIII, IX, X compared with Group II. * P < 0.00, # P < 0.01, @ P < 0.05.

The number of different types of sperm abnormalities is shown in both Table.2. & Figure.1. The percentage of abnormal sperm is shown in Table.3. and Figure.2. and the total number of sperm count is represented in Table.3. and Figure.3. Amorphous head, Headless Sperm, Tailless Sperm, Bent at the cephalocaudal region, Bent tail, and other abnormalities such as two-tailed sperms were all taken into account while looking for sperm abnormalities (Figure.4.). A few aberrant sperms per thousand sperms were found in both the vehicle control and D-P solely treatment groups. On the alternative side, the positive control, DOX, demonstrated a significant rise in the abnormalities in sperm shape. The D-P and DOX groups exhibited a significant ($P<0.001$) decline in sperm abnormalities in a dose-dependent manner as compared to the DOX-only treated group. Both the vehicle control and D-P alone treated groups of mice had normal sperm counts. However, compared to the vehicle control mice, animals treated with DOX had a significantly ($P<0.001$) reduced sperm count. When supplemented with DOX, D-P significantly ($P<0.001$) raised sperm count in a dose-dependent manner.

Genotoxic studies are useful for understanding the extent of DNA damage caused by medication. Genotoxic substances can impair a cell's genetic makeup [21][22]. DOX, a genotoxic agent is possible inducers of sperm cell morphology changes because they can affect the normal events of gametogenesis [23]. In support of the earlier result, the current investigation revealed that DOX treatment, by its capacity to trigger oxidative stress and inflammatory activity, significantly increased sperm shape abnormalities and lowered sperm count [24]. D-P did not exhibit any abnormalities in the shape of sperm and sperm count when tested for genotoxicity. From the Table.2. & Table.3. and Figure.2. & Figure.3., it is also revealed that administration of DOX resulted in an abnormal reduction in the sperm shape abnormality and an increase in sperm counts. While our findings explicitly showed that pre-administration of D-P with DOX decreased genotoxicity in germ cells caused by DOX in mice, as demonstrated by reduced sperm shape defects and improved sperm count.

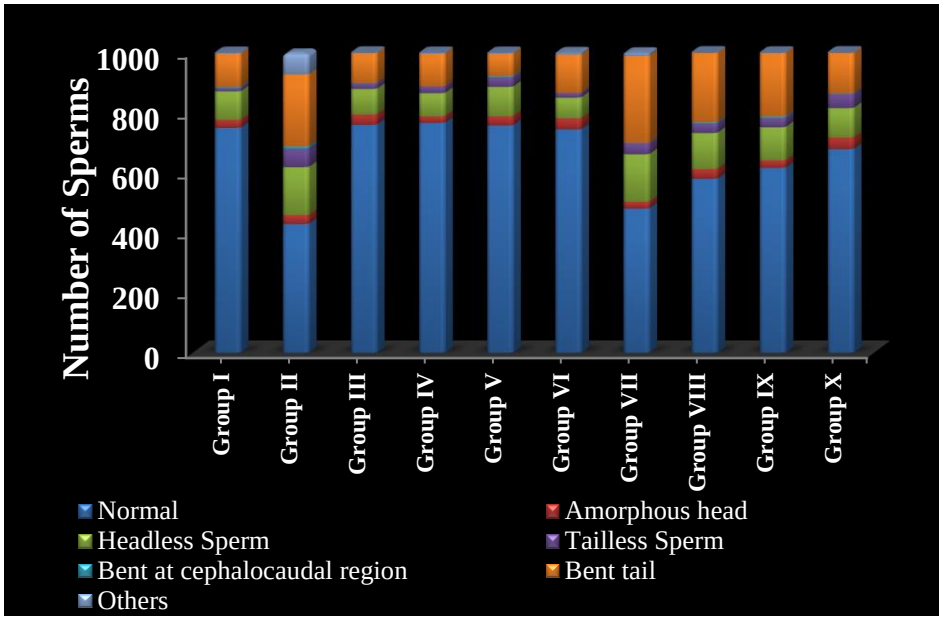


Figure 1: Histogram - Number of Sperm abnormalities that occurred in treated groups of mice

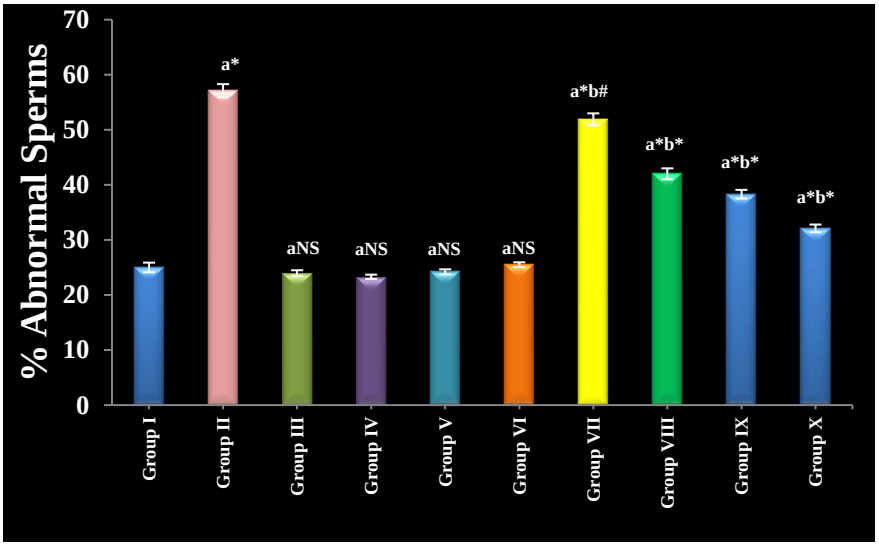


Figure 2: Histogram - Percentage of abnormal sperms

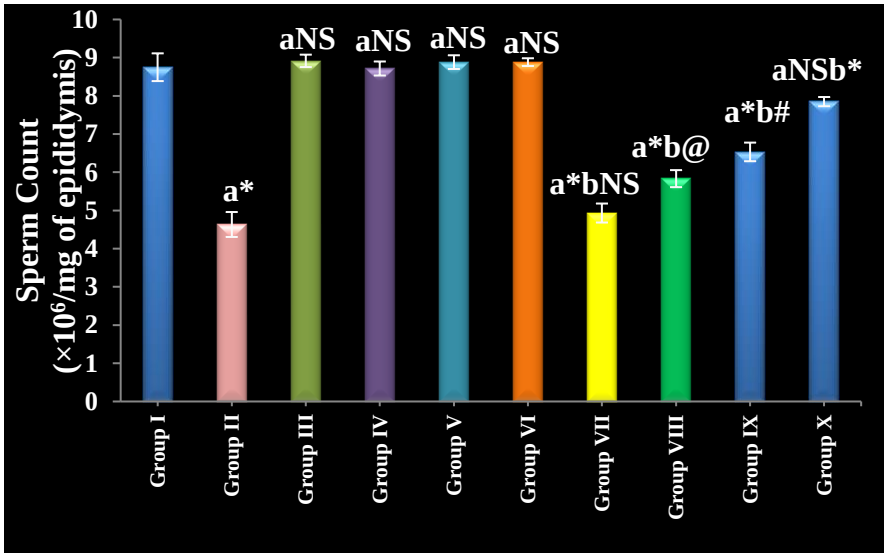


Figure 3: Histogram - Total Number of Sperm Count

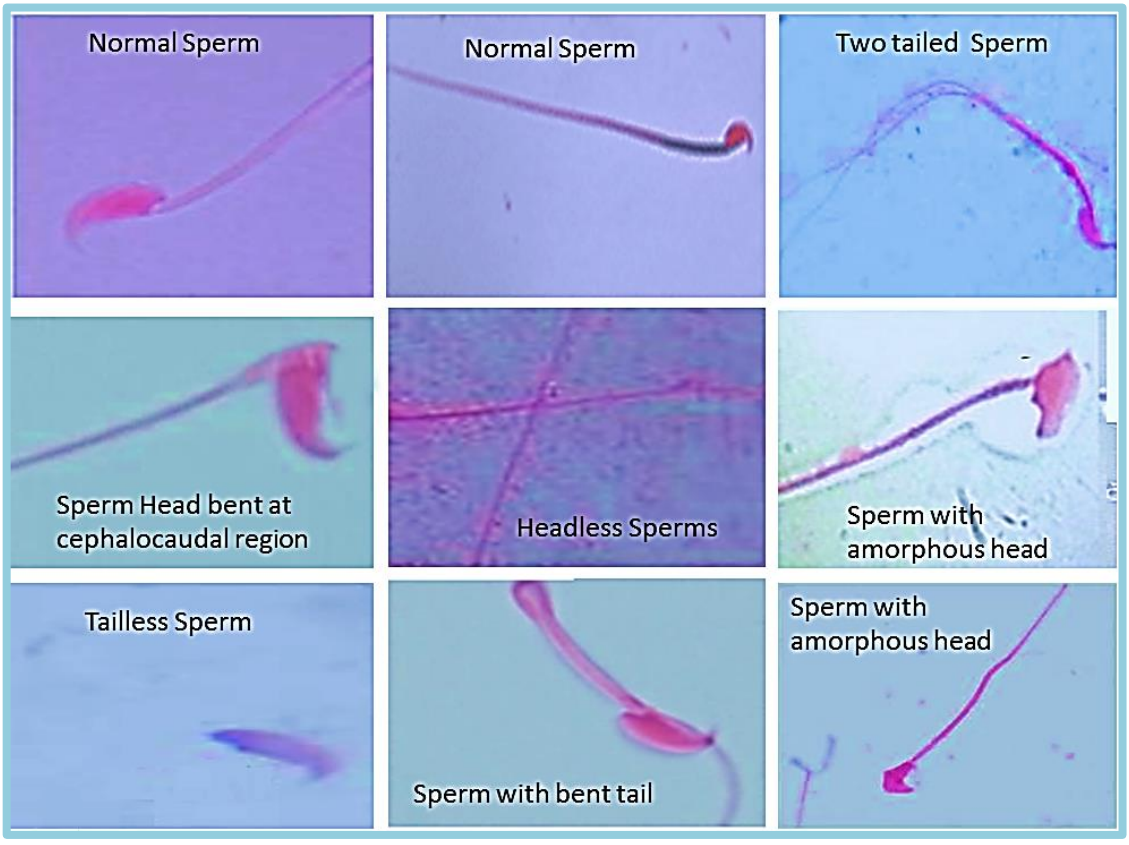


Figure 4: Abnormal Sperms in the treated groups of mice

Conclusion:

D-P has a genoprotective effect against DOX-induced genotoxicity in germ cells. The genotoxic evaluation of D-P revealed that it did not induce any genotoxic effects. The antioxidant and anti-inflammatory properties of D-P would be the foremost reason for its genoprotective effect.

Acknowledgment:

The author thanks the authorities of Adhiparasakthi College of Pharmacy for providing the necessary facilities to do this research.

List of abbreviations:

DOX – Doxorubicin

D-P – D–Pinitol

Informed consent:

The Institutional Animal Ethics Committee (IAEC) of Adhiparasakthi College of Pharmacy (Reg. No. 409/PO/Re/S/01/CPCSEA) approved the experimental protocol for *in vivo* chromosomal aberration assay. The approval number was APCP/IAEC/2019-2020/1.

References:

1. Jemal, A., Bray, F., Center, MM., Ferlay, J., *et al.* (2011). Global cancer statistics. *CA: A Cancer Journal for Clinicians*. 61(2): 69–90. doi:10.3322/caac.20107
2. McKnight, JA. (2003). Principles of Chemotherapy. *Clinical techniques in small animal practice*. 18(2): 67-72.
3. Gewirtz, DA. (1999). A critical evaluation of the mechanisms of action proposed for the antitumor effects of the anthracycline antibiotics adriamycin and daunorubicin. *Biochemical Pharmacology*. 57: 727-741.
4. Pommier, Y., Leo, E., Zhang, H., Marchand, C. (2010). DNA Topoisomerases and Their Poisoning by Anticancer and Antibacterial Drugs. *Chemistry & Biology*.17(5):421-433. doi: 10.1016/j.chembiol.2010.04.012
5. Hajra, S., Patra, AR., Basu, A., Bhattacharya, S. (2018). Prevention of doxorubicin (DOX)-induced genotoxicity and cardiotoxicity: Effect of plant derived small molecule indole-3-carbinol (I3C) on oxidative stress and inflammation. *Biomedicine & Pharmacotherapy*. 101: 228-243.
6. Wang, L., Chen, Q., Qi, H., Wang, C., *et al.* (2016). Doxorubicin-Induced Systemic Inflammation Is Driven by Upregulation of Toll-Like Receptor TLR4 and Endotoxin Leakage. *Cancer Research*. 76(22): 6631-6642. doi: 10.1158/0008-5472.CAN-15-3034
7. Rengarajan, T., Nandakumar, N., Rajendran, P., Haribabu, L., *et al.* (2014) D-Pinitol Promotes Apoptosis in MCF-7 Cells via Induction of p53 and Bax and Inhibition of Bcl-2 and NF-κB. *Asian Pacific Journal of Cancer Prevention*. 15(4): 1757-1762.
8. Jayasooriya, RGPT., Kang, C-H., Park, SR., Choi, Y-H., *et al.* (2015). Pinitol Suppresses Tumor Necrosis Factor-α-Induced Invasion of Prostate Cancer LNCaP Cells by Inhibiting

Nuclear Factor-κB-Mediated Matrix Metalloproteinase-9 Expression. *Tropical Journal of Pharmaceutical Research.* 14(8): 1357-1364. doi: 10.4314/tjpr.v14i8.6.

9. Poongothai, G., Sripathi, SK. (2013) A review on insulinomimetic Pinitol from plants. International *Journal of Pharmacy and Biological Sciences.* 4(2): 992-1009.
10. Streeter JG. (2001) Simple partial purification of D-Pinitol from Soybean leaves. *Crop Science.* 41: 1985-1987.
11. Rengarajan, T., Balasubramanian, MP., Rajendran, P., Nandakumar, N., *et al.* (2014). Free radical scavenging and antioxidant activity of D-pinitol against 7, 12- Dimethylbenz(a) Anthracene induced breast cancer in Sprague Dawley rats. *Asian Pacific Journal of Tropical Disease*. 4(5): 384-390. doi:10.1016/S2222-1808(14)60592-2
12. López-Domènech, S., Bañuls, C., de Marañón, AM., Abab-Jiménez, Z., *et al.* (2018) Pinitol alleviates systemic inflammatory cytokines in human obesity by a mechanism involving unfolded protein response and sirtuin 1. *Clinical Nutrition*. 37: 2036-2044. doi: 10.1016/j.clnu.2017.09.015.
13. Rengarajan, T., Jagadeesan, AJ., Balamurugan, A., Balasubramanian, MP., *et al.* (2011). Chemotherapeutic potential of D-Pinitol against 7, 12-Dimethylbenz (a) anthracene (DMBA) induced mammary carcinoma in Sprague Dawley Rats. *International Journal of Pharma and Bio Sciences.* 2(4): 232-241.
14. Kim, J-I., Kim, JC., Kang, M-J., Lee, M-S., *et al.* (2005). Effects of pinitol isolated from Soybeans on glycaemic control and cardiovascular risk factors in Korean patients with type II diabetes mellitus: a randomized controlled study. *European Journal of Clinical Nutrition*. 59(3): 456-458. doi:10.1038/sj.ejcn.1602081.
15. Srivastava, K., Tiwari, M., Dubey, A., Dubey, A. (2020) D-Pinitol - A Natural Phytomolecule and its Pharmacological effect. *International Journal of Pharmaceutical and Life Sciences*. 11(5): 6609-6623.
16. Vasaikar, N., Mahajan, U., Patil, KR., Suchal, K., *et al.* (2018). D-Pinitol attenuates cisplatin-induced nephrotoxicity in rats: Impact on Pro-inflammatory cytokines. *Chemico-Biological Interactions.* 290: 6–11. doi:10.1016/j.cbi.2018.05.003
17. http://cpcsea.nic.in/WriteReadData/userfiles/file/SOP_CPCSEA_inner_page.pdf
18. Navarro, JA., Decara, J., Medina-Vera, D., Tovar, R., Suarez, J., Pavón, J., *et al.* (2020). D-Pinitol from *Ceratonia siliqua* Is an Orally Active Natural Inositol That Reduces Pancreas Insulin Secretion and Increases Circulating Ghrelin Levels in Wistar Rats. *Nutrients.* 1452(7): 1-22.

19. Padmanabhan, S., Tripathi, DN., Vikram, A., Ramarao, P., *et al.* (2009). Methotrexate-induced cytotoxicity and genotoxicity in germ cells of mice: Intervention of folic and folinic acid. *Mutation Research*. 673(1): 43-52.
20. Sharma, R., Singh, S., Singh, GD., Khajuria, A., *et al.* (2009). *In vivo* genotoxicity evaluation of a plant based antiarthritic and anticancer therapeutic agent Boswelic acids in rodents. *Phytomedicine*. 16:1112–1118. doi:10.1016/j.phymed.2009.06.009
21. Baidya, M., Manna, K., Maji, HS., Mandal, SK., (2022). *In Vivo* Evaluation of Genotoxic Effects of Sivanar Amirtham Formulation on Rats Using Micro Nucleus Assay. *Research Journal of Pharmacy and Technology*. 15(11):5017-0. doi: 10.52711/0974-360X.2022.00843
22. Sumanth, M., Swetha, S., Narasimharaju, K., Anusha, Natesh, T.S., *et al.* (2011). Genotoxicity Testing of Lipovedic- A Polyherbal Anti-hypercholesterolemic Drug. *Research Journal of Pharmacy and Technology*. 4(8): 1189-1192.
23. Shinoda, K., Mitsumori, K., Yasuhara, K., Uneyama, C., *et al.* (1999). Doxorubicin induces male germ cell apoptosis in rats. *Archives of Toxicology*. 73(4-5): 274–281. doi:10.1007/s002040050617.
24. Takahashi, H., Tainaka, H., Umezawa, M., Takeda, K., Tanaka, H., Nishimune, Y., *et al.* (2011). Evaluation of testicular toxicology of doxorubicin based on microarray analysis of testicular specific gene expression. *The Journal of Toxicological Sciences*. 36(5):559-67. doi:10.2131/jts.36.559.

ADAPTOGENIC ACTIVITY OF D-PINITOL AGAINST DIVERSE STRESSORS INDUCED IN SWISS ALBINO MICE

Vinciya T*[1], Sudha M[1], Dhanalakshmi A[2],

Noorul Alam I[3], Aishwarya P J[1] and Harikrishnan N[4]

[1]Department of Pharmacology, Faculty of Pharmacy, Dr. M.G.R. Educational and Research Institute (Deemed to be University), Chennai, Tamil Nadu, India.

[2]Department of Pharmacology, Mohammad Satak AJ college of Pharmacy, Chennai

[3]Department of Pharmaceutics, College of Pharmacy,
Mother Theresa Post Graduate and Research Institute of Health Sciences, Puducherry.

[4]Department of Pharmaceutical Analysis, Faculty of Pharmacy, Dr. M.G.R. Educational and Research Institute (Deemed to be University), Chennai, Tamil Nadu, India.

*Corresponding author E-mail: vinciya.pharm@drmgrdu.ac.in

Abstract:

D-Pinitol is evaluated for its adoptogenic activity/anti-stress activity through forced swimming test along with post-motor performance, tail suspension test, and elevated maze test. Four groups consisting of six Swiss albino mice (20–30 g each) were used for this investigation. Mice without administering any drug were kept as Control. Standard (Diazepam 2 mg/kg i.p.), test drug low dose (D-Pinitol 100 mg/kg) and test drug high dose (D-Pinitol 400 mg/kg) were administered to mice for assessing adaptogenic activity. Compared with the control group, all doses of D-Pinitol showed significant reduction in the immobility time for both tail suspension test and forced swimming test, increased swimming endurance time, and improved post-motor performance, including spontaneous motor activity and Rota rod falling time. In comparison to the control group, D-Pinitol (100 mg/kg and 400 mg/kg) significantly increased the amount of time that was spent in the open arm present in the Elevated Plus Maze. The previous study found that stress results in the production of free radicals. In animals, stress also causes hyperlipidemia, hypoglycemia, and a rise in serum cortisol levels. Based on previous research, capacity of D-Pinitol as an antioxidant, and lowering abnormal blood levels of glucose, cortisol, and cholesterol can be utilized to correct various pathological conditions aforementioned to treat and prevent stress. This study intended to show that D-Pinitol can prevent stress disorders at in vivo levels because it showed adaptogenic activity or anti-stress effect.

Keywords: D-Pinitol, Diazepam, Adaptogenic Activity, Forced Swimming Test, Tail Suspension Test, Elevated Maze Test.

Introduction:

Adaptogens are stress-response modulators which improve a person's generalised resilience to stressful conditions by enhancing the capacity for survival and adaptation (1)(2). Adaptogens fall into two primary categories. Plant adaptogens fall under the first category, whereas synthetic adaptogens (also known as actoprotectors) fall under the second category (3). According to scientific evidence, high or continuous psychological stress can cause persistent disorders of neurological function, some of which entail the destruction of neuronal cells. Recent research is currently underway exploring the biological mechanisms of psychological stress activates or inhibits. In psychological stress, mechanism of stress induction involves activation of hypothalamic-pituitary-adrenal axis leading to release of excessive cortisol and occurrence of inflammation in the brain because of cytokines release leading to oxidative stress (4).

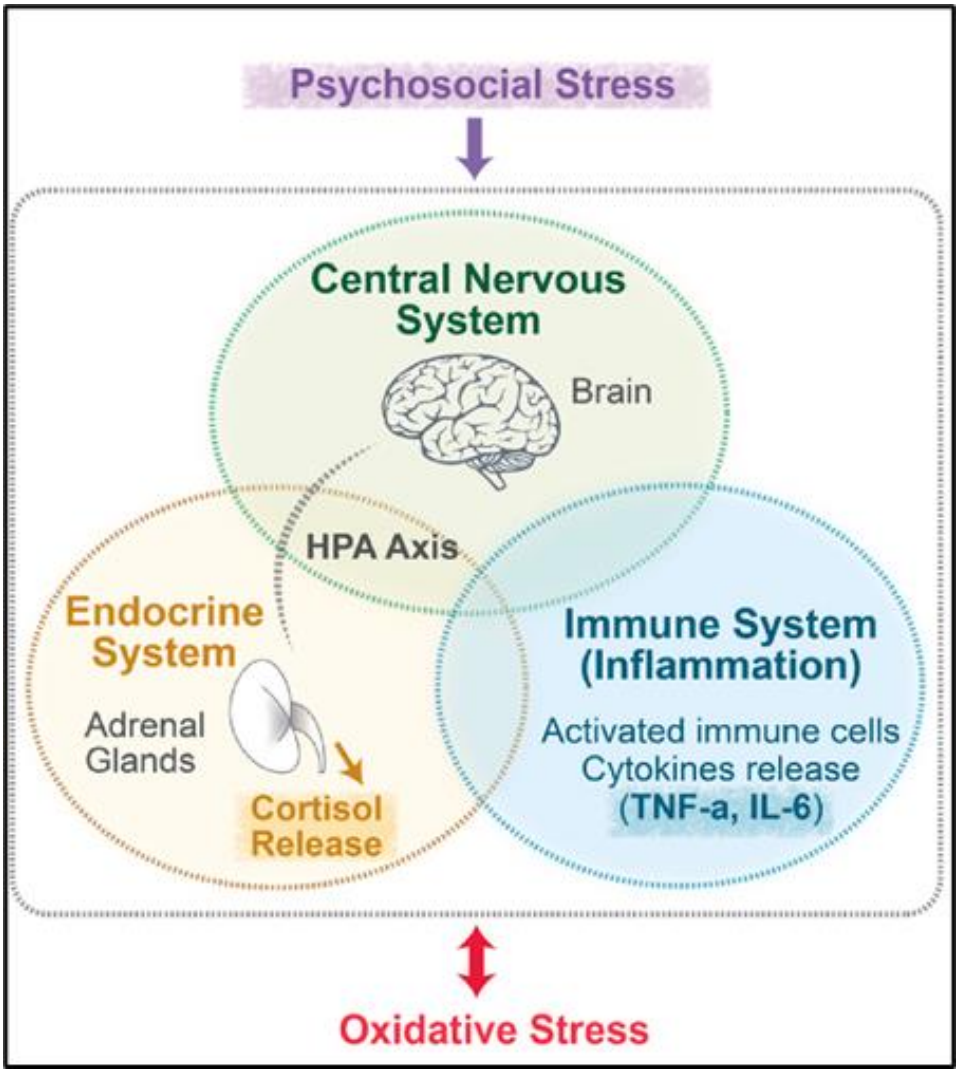


Figure 1: Psychological stress induced inflammatory oxidative stress (5)

According to previously published research, people with psychological issues caused by stress could benefit from antioxidant supplement treatment given as an adjunct to conventional therapy. Therefore, antioxidant supplement therapy is beneficial for patients with stress-induced oxidative stress as an adjuvant therapy or therapy (6).

D-Pinitol, 3-O-methyl ether of D-Chiroinositol, is occurring in various plant sources prevalently including *Gliricidia sepium*, *Bougainvillea spectabilis*, *Glycine max* L Merr., and *Tamarindus Indica* Linn (7). D-Pinitol has been documented in more than 30 medical uses, including anti-diabetic, insulin-regulator, anti-Alzheimer's, anti-cancer, antioxidant, anti-inflammatory properties and hepatoprotective properties (8). As a result, this research study aimed to assess the adaptogenic activity of D-Pinitol using Swiss Albino mice.

Material and Methods:

***In vivo* adaptogenic activity:**

Animal care and handling:

Both sex of Swiss Albino Mice (Weight: 25–30 g) were housed with a 12-hour light/dark cycle in cages. The animals were acclimated in cages and provided with a clean and controlled environment in accordance with the Committee for Control and Supervision of Experiments on Animals (CCSEA) specifications before the experiment began (9). Dose selections for Diazepam and D-Pinitol were based on Ghias, M, *et al.* (10) and Navaaro *et al*. respectively (11).

Materials required:

D-Pinitol (TCI Chemicals), Diazepam injection (NEON Laboratories Limited), Glass cylinders 2 numbers (height 25 cm, diameter 10 cm), Tap Water, Burette Stand with clamp, Micropore Adhesive tape, and Elevated Plus Maze Model.

Methodology:

Table 1: Treatment Protocol

Group	Labelled	Treatment
I	Control	-
II	Standard Drug	Diazepam (2 mg/Kg) i.p.
III	Test Drug 1– Low Dose	D-Pinitol (100 mg/Kg) p.o.
IV	Test Drug 2 – High Dose	D-Pinitol (400 mg/Kg) p.o.

I. Forced swimming endurance test and post-swimming motor function test:

The untrained mice for swimming were used in the forced swimming test. The glass cylinder with the height of 25 centimeter and with the diameter of 10 centimeter was poured with

10 cm of water having normal temperature. Each mouse was placed inside a glass cylinder and to measure the period of time during which the animals were mobile and immobile, they were left in the cylinder for 6 minutes. The final 4 minutes of the 6 minutes testing session were used to calculate the total duration of immobility. The mouse was believed to be immobile when it stopped its attempts to move and remained unmoved within the water, moving only to maintain its head above the surface. Antistressor will shorten the period of immobility (12). Next, the mice were allowed to swim until they became too exhausted, and the time it took for them to drown was recorded (13). The animals were taken away and given about five minutes to recover and dry. On a Rota rod, the animals were evaluated for muscular coordination, and the duration of time spent on the rod was recorded. For about ten minutes, they were then monitored for spontaneous motor activity in a photoactometer (14).

II. Tail suspension test:

Individual animals were suspended 50 cm from the bottom on the burette stand clamp with micropore adhesive tape (approximately 1 cm) at the tail end. For a total of six minutes, mice were suspended in the stand. The last 4 minutes of the test were used to assess the duration of immobility. Only when mice are hanging passively and without any movement, they are believed to be immobile. In these experiments, anti-stressor reduces the immobility of the mice (15).

III. Elevated plus maze test:

The elevated plus maze was made up of two open arms (each measuring 50 cm by 10 cm) intersected by two closed arms (each measuring 50 cm by 10 cm by 40 cm). A center square (10 cm by 10 cm) joined the arms together. The maze was constructed with a height of 70 cm. Following sixty minutes after administration of the medication, each mouse was placed separately in the middle of the maze with their heads toward the open arm. Subsequent behavioral parameters were taken for a duration of five minutes. The length of time spent in the open arm was recorded (16).

Statistical analysis:

Statistics were evaluated to be significant at P values under 0.05 ($P<0.05$). One-way ANOVA followed by Tukey test as *post hoc* test was performed statistically using GraphPad Prism software version 8.01 for this research.

Results and Discussion:

Table 2: Effect of D-Pinitol on mice in immobility time in swimming endurance test

Groups	Labelled/Dose	Forced Swimming Endurance Test
		Immobility Time (In Seconds)
I	CONTROL	139.2 ± 19.33
II	DIAZEPAM 2 mg/Kg	65.17 ± 9.131 ***
III	D-PINITOL 100 mg/Kg	94.33 ± 15.02 ***
IV	D-PINITOL 400 mg/Kg	108 ± 3.742 ***

N=6; mean ± S.E.M; Where Group II,

Group III & Group IV were compared with Group I; *p < 0.05, **p < 0.01, ***p < 0.001

During the forced swimming test, the control animals continued to be immobile for the majority of the time. The immobility time of control, standard (Diazepam 2 mg/Kg) and test (D-Pinitol 100 mg/kg and D-Pinitol 400 mg/Kg) were depicted in Table.2 and Figure.2.

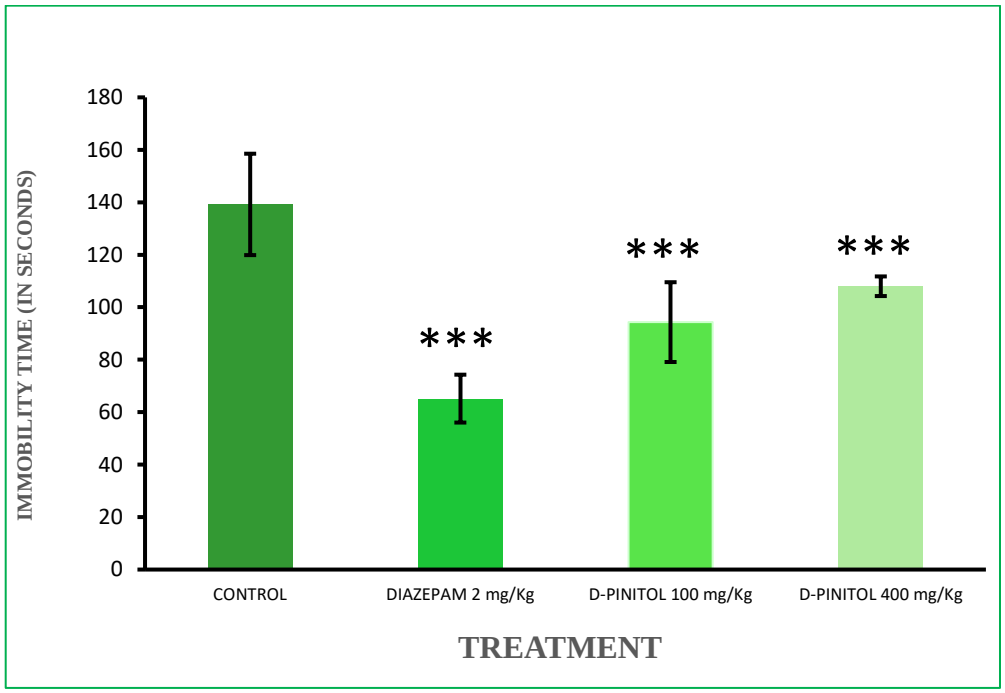


Figure 2: Histogram - Effect of D-Pinitol on mice in immobility time in swimming endurance test

Table 3: Effect of D-Pinitol on mice in swimming endurance in minutes

Groups	Labelled/Dose	Duration Of Swimming Endurance (Till Drowning) In Minutes
I	Control	7.061 ± 2.02
II	Diazepam 2 mg/Kg	14.30 ± 4.293 **
III	D-Pinitol 100 mg/Kg	21.3 ± 1.981 **
IV	D-Pinitol 400 mg/Kg	21.02 ± 1.35 **

N=6; mean ± S.E.M; Where Group II,

Group III & Group IV were compared with Group I; *p < 0.05, **p < 0.01, ***p < 0.001

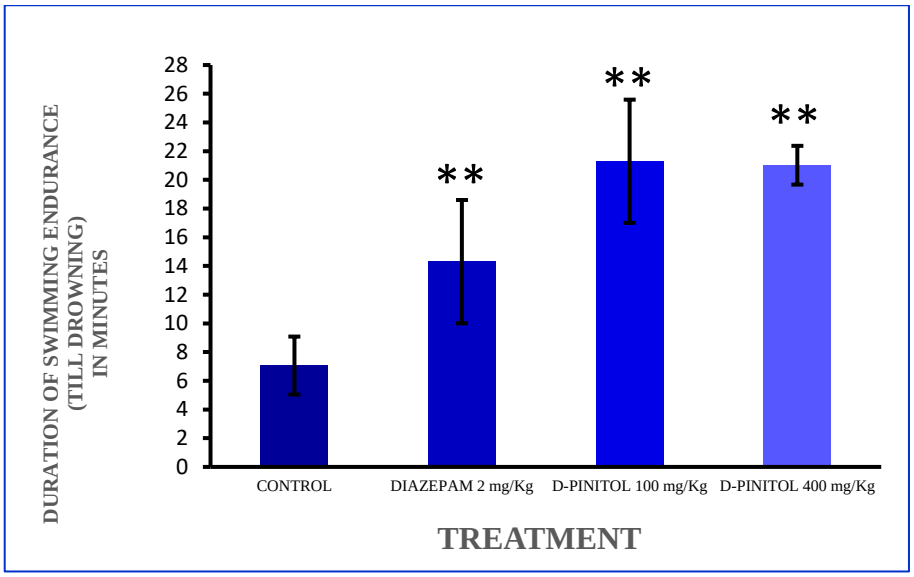


Figure 3: Histogram - Effect of D-Pinitol on mice in swimming endurance in minutes

Table 4: Effect of D-Pinitol on mice in Rota Rod falling off time

Groups	Labelled/Dose	Rota Rod Falling Off Time (In Seconds)
I	Control	12.5 ± 1.871
II	Diazepam 2 mg/Kg	45.5 ± 6.565 ***
III	D-Pinitol 100 mg/Kg	23.67 ± 4.412 **
IV	D-Pinitol 400 mg/Kg	80.67 ± 8.618***

N=6; mean ± S.E.M; Where Group II,

Group III & Group IV were compared with Group I; *p < 0.05, **p < 0.01, ***p < 0.001

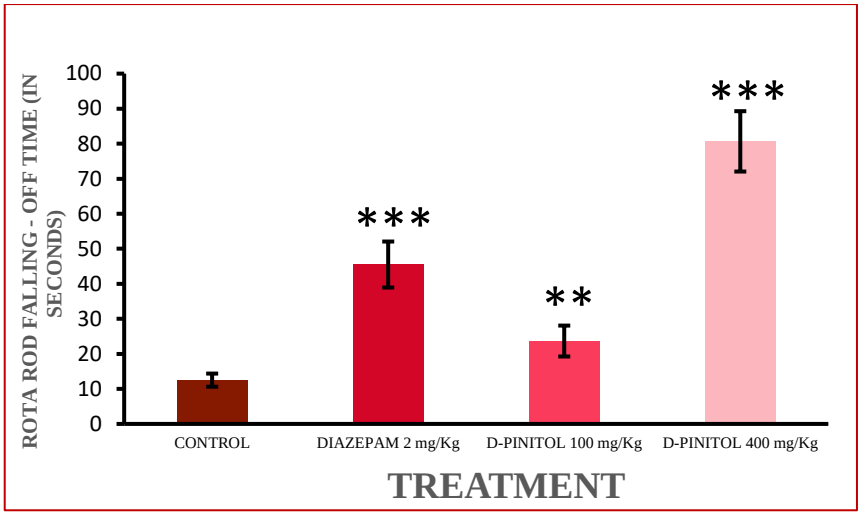


Figure 4: Histogram - Effect of D-Pinitol on mice in Rota Rod falling off time post-swimming motor function test

Table 5: Effect of D-Pinitol on mice in Spontaneous Motor Activity done (Photoactometer) in post-swimming motor function test

Groups	Labelled/Dose	Spontaneous Motor Activity Scores (In Minutes)
I	Control	83 ± 5.215
II	Diazepam 2 mg/Kg	181 ± 7.321 ***
III	D-Pinitol 100 mg/Kg	202.1 ± 5.636 ***
IV	D-Pinitol 400 mg/Kg	238.7 ± 23.47 ***

N=6; mean ± S.E.M; Where Group II,

Group III & Group IV were compared with Group I; *p < 0.05, **p < 0.01, ***p < 0.001

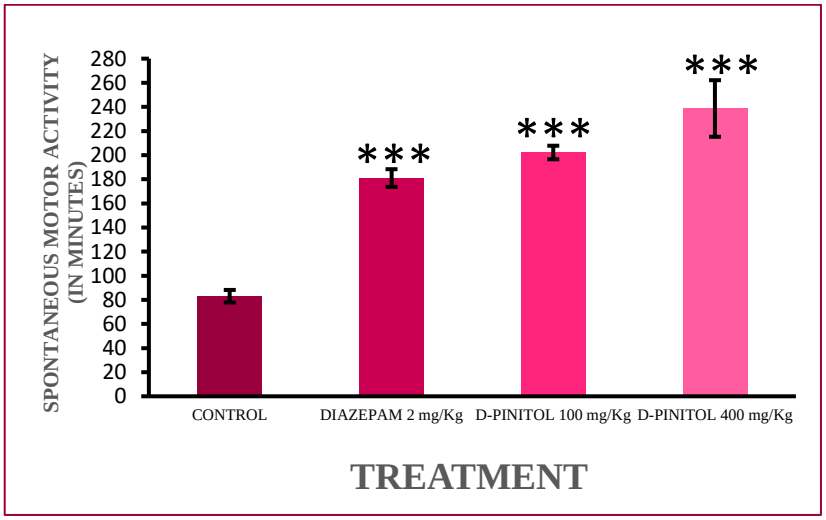


Figure 5: Histogram - Effect of D-Pinitol on mice in Spontaneous Motor Activity done (Photoactometer) in post-swimming motor function test

Table 6: Effect of D-Pinitol on mice in immobility time in Tail suspension test

Groups	Labelled/Dose	Tail Suspension Test
		Immobility Time in Seconds
I	Control	174.8 ± 9.13
II	Diazepam 2 mg/Kg	105 ± 26.89 ***
III	D-Pinitol 100 mg/Kg	114.5 ± 30.41 ***
IV	D-Pinitol 400 mg/Kg	90.67 ± 15.16 ***

N=6; mean ± S.E.M; Where Group II,

Group III & Group IV were compared with Group I; *p < 0.05, **p < 0.01, ***p < 0.001

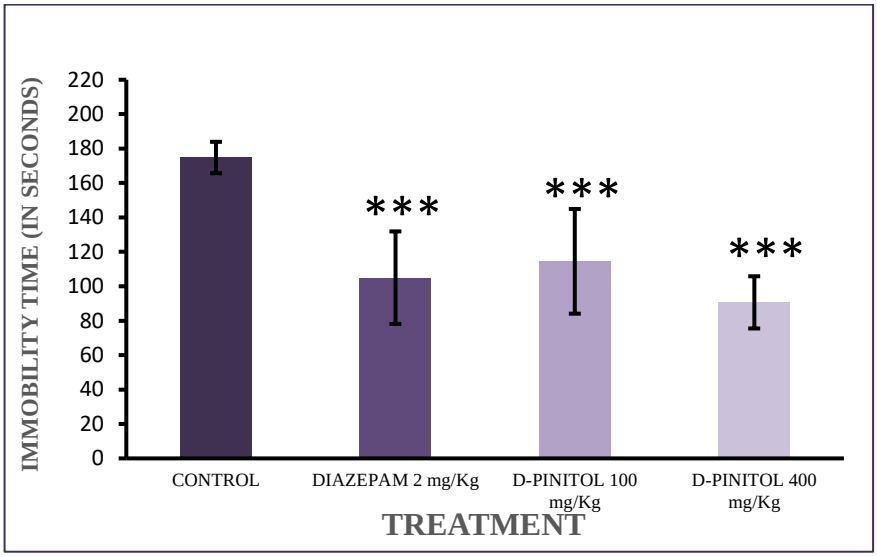


Figure 6: Histogram - Effect of D-Pinitol on mice in immobility time in Tail suspension test

Table 7: Effect of D-Pinitol on mice in elevated plus maze model

Groups	Labelled/Dose	Time Spent in Open Arm (In Seconds)
I	Control	75 ± 4
II	Diazepam 2 mg/Kg	257.8 ± 18.65***
III	D-Pinitol 100 mg/Kg	165.2 ± 2.787 ***
IV	D-Pinitol 400 mg/Kg	184.3 ± 6.976***

N=6; mean ± S.E.M; Where Group II, Group III & Group IV were compared with Group I;

*p < 0.05, **p < 0.01, ***p < 0.001

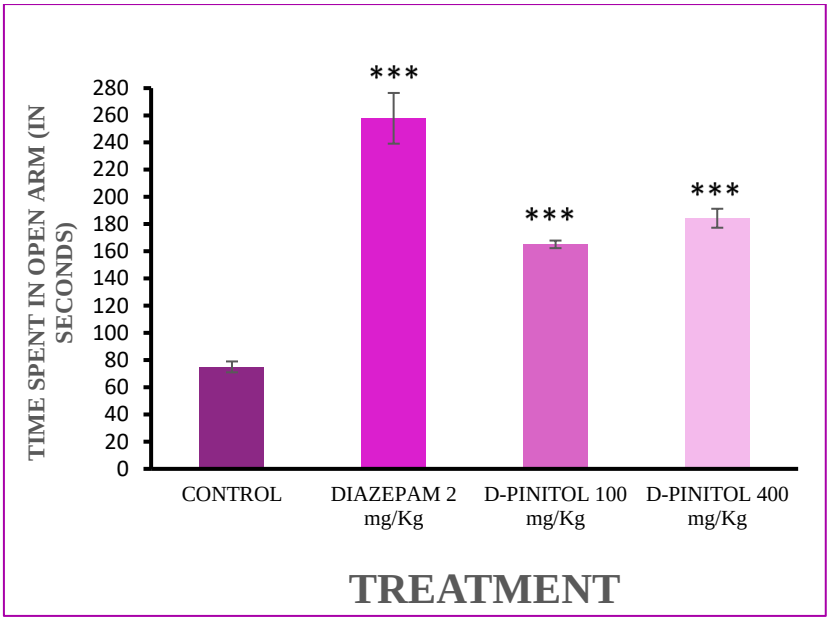


Figure 7: Histogram - Effect of D-Pinitol on mice in Elevated plus Maze Model

The immobility time of the standard diazepam and test drugs, viz., D-Pinitol in low and high doses were significantly (***P<0.001) reduced compared to the control. Diazepam, D-Pinitol 100 mg/Kg, and D-Pinitol 400 mg/Kg significantly (**P<0.01) lengthened the time that mice spent swimming before drowning compared to control animals (Table 3 and Figure 3). Hence, mice pre-treated with D-Pinitol in both doses were shown to have longer swimming endurance. The duration spent on the Rota rod was considerably prolonged by Diazepam (***P<0.001), D-Pinitol 100 mg/Kg (**P<0.01), and D-Pinitol 400 mg/Kg (***P<0.001) compared with that of control. These findings are shown in Table 4 and Figure 4. The photoactometer measurement for spontaneous motor activity was significantly (***P<0.001) improved by diazepam, D-Pinitol 100 mg/Kg, and D-Pinitol 400 mg/Kg (Table 5 and Figure 5) compared with that of control. In the tail suspension test, which was depicted in Table 6 and Figure 6, Diazepam and both of the D-Pinitol test doses significantly (***P<0.001) reduced the amount of time spent immobile compared to the control. In elevated plus maze test, mice receiving the standard dose of diazepam and D-Pinitol (100 mg/Kg and 400 mg/Kg) increased their time spent in the open arm significantly (***P<0.001) compared to the control group. These findings are given in Table 7 and Figure 7.

Stress, which is brought on by free radicals, is the root cause of many human disorders. Continuous stress raises free radical levels, which causes an aberrant physiological condition, the emergence of psychological dysfunction, and the decline in cognitive performance (17). The other pathological findings involved in stress was activation of hypothalamic-pituitary-adrenal

axis leading to the release of cortisol (14), which subsequently causes the mobilization of fats, carbohydrates, and lipids from storage and elevates blood sugar, triglyceride, and cholesterol levels (18). In earlier studies conducted by Rengarajan *et al.*, the effect of D-Pinitol in reducing free radical scavenging and its antioxidant capacity were proved (19). D-Pinitol has previously been shown to be able to lower blood sugar levels in diabetic animals by changing the levels of the hormones that regulate blood sugar, including glucagon, and insulin and to reduce elevated plasma level of cortisol (20). Because of its aforementioned properties, D-Pinitol may be accountable for the observed anti-stress/adaptogenic action in this study using diverse stressor models in mice.

Conclusion:

The prospective behavioral effects of D-Pinitol were examined in this study using a variety of acute stress methods. D-Pinitol (100 mg/Kg and 400 mg/Kg) has considerably proved its capacity to stop the changes brought on by stress when administered to mice. Therefore, it can be said and inferred from all stressor studies that D-Pinitol possesses adaptogenic property (antistress effect) and thereby gives protection for stress induction. D-Pinitol's adaptogenic action was possibly due to its antioxidant property and its ability to ameliorate abnormally elevated plasma glucose, cortisol, and fatty acid levels.

Acknowledgment:

The authors thank the authorities of Faculty of Pharmacy, Dr.M.G.R. Educational and Research Institute, Deemed to be University, Chennai, Tamil Nadu for providing the necessary facilities to do this research.

Informed consent:

The Institutional Animal Ethics Committee (IAEC) of A.C.S. Medical College, Dr. M.G.R. Educational and Research Institute (Deemed to be University), approved the experimental protocol. The approval number was VI/IAEC/Dr MGR/2053/PO/ReBi/S/19/CPCSEA/28.01.2023/05.

References:

1. Brekhman, II., Dardymov, IV. (1969). New substances of plant origin increase nonspecific resistance. *Annual review of pharmacology*. 9(1):419-430.
2. Wagner, H., Nörr, H., Winterhoff, H. (1994). Plant adaptogens. *Phytomedicine*. 1(1): 63-76.
3. Todorova, V., Ivanov, K., Delattre, C., Nalbantova, V., Karcheva-Bahchevanska, D., Ivanova, S. (2021). Plant Adaptogens-History and Future Perspectives. *Nutrients.* 13(8):2861. doi: 10.3390/nu13082861.

4. Kim, E., Zhao, Z., Rzasa, JR., Glassman, M., Bentley, WE., Chen, S., *et al.* Association of acute psychosocial stress with oxidative stress: Evidence from serum analysis. *Redox Biology*. 2021; 47.
5. Hayashi, T. (2015). Psychological stress and cellular stress. *Psychiatry Clin Neurosci*. 69:179-191. https://doi.org/10.1111/pcn.12262.
6. Gautam, M., Agrawal, M., Gautam, M., Sharma, P., Gautam, AS., Gautam, S. (2012). Role of antioxidants in generalised anxiety disorder and depression. *Indian J Psychiatry*. 54(3):244-7. doi: 10.4103/0019-5545.102424.
7. Sripathi, SK., Poongothai, G. (2013). A review on insulinomimetic Pinitol from plants. *Int J Pharm Bio Sci*. 4(2): 992-1009.
8. Azab, A. (2022). D-Pinitol—Active Natural Product from Carob with Notable Insulin Regulation. *Nutrients*. 14(7):1453.
9. http://cpcsea.nic.in/WriteReadData/userfiles/file/SOP_CPCSEA_inner_page. pdf
10. Ghias, M., Shah, SWA., Al-Joufi, FA., Shoaib, M., Shah, SMM., Ahmed, MN., Zahoor, M. (2022). *In Vivo* Antistress Effects of Synthetic Flavonoids in Mice: Behavioral and Biochemical Approach. *Molecules*. 27(4):1402. https://doi.org/10.3390/molecules27041402
11. Navarro, JA., Decara, J., Medina-Vera, D., Tovar, R., *et al.* (2020). D-Pinitol from *Ceratonia siliqua* Is an Orally Active Natural Inositol That Reduces Pancreas Insulin Secretion and Increases Circulating Ghrelin Levels in Wistar Rats. *Nutrients*. 1452(7): 1-22.
12. Wośko, S., Serefko, A., Socała, K., Szewczyk, B., Wróbel, A., Nowak, G., *et al.* (2014). An anti-immobility effect of spermine in the forced swim test in mice. *Pharmacol Rep.* 66(2):223-7. doi: 10.1016/j.pharep.2013.10.002.
13. Kannur, DM., Hukkeri, VI., Akki, KS. (2006). Adaptogenic activity of *Caesalpinia bonduc* seed extracts in rats. *J Ethnopharmacol*. 108(3):327-31. doi: 10.1016/j.jep.2006.05.013.
14. Duraisami, R., Mohite, VA. (2010). Anti stress, adaptogenic activity of standardized dried fruit extract of Aegle marmelos against diverse stressors. *Asian J Pharm Clin Res*. 3(4): 11-13.
15. Ansari, I., Sorte, RS. (2018). Evaluation of Anti-Stress activity of Ethanolic Extracts of *Terminalia Catappa* L. in Swiss Albino Mice. *Asian Journal of Pharmaceutical and Clinical Research.* 11(6): 253-257. doi:10.22159/ajpcr.2018.v11i6.25046.

16. Tiwari, N., Mishra, A., Bhatt, G., Chaudhary A. (2014). Anti Stress Activity (*in-vivo*) of Forskolin Isolated from Coleus forskohlii. *Int J Pharm Phytopharmacol Res*. 4 (3): 201-204.
17. Singh, S., Upadhyay, A., Sirbaiya, AK. (2021). Neuropharmacological Screening, Anti-Stress Activity, And Toxicity Studies of Standardized Extract of The Seeds of *Celastrus Paniculatus* Willd. *Asian Journal of Pharmaceutical and Clinical Research*. 14(11):52-56.
18. Nade, VS., Kawale, LA., Naik, RA., Yadav, AV. (2009). Adaptogenic effect of *Morus alba* on chronic footshock-induced stress in rats. *Indian J Pharmacol*. 41: 246 -51.
19. Rengarajan, T., Balasubramanian, MP., Rajendran, P., Nandakuma,r N., *et al.* (2014). Free radical scavenging and antioxidant activity of D-pinitol against 7, 12- Dimethylbenz(a) Anthracene induced breast cancer in Sprague Dawley rats. *Asian Pac J Trop Dis*. 4(5): 384-390. doi:10.1016/S2222-1808(14)60592-2.
20. Navarro, JA., Díaz, C., Decara, J., Medina-Vera, D., Lopez-Gambero, AJ., Suarez, J., *et al.* (2022). Pharmacokinetics and Endocrine Effects of an Oral Dose of D-Pinitol in Human Fasting Healthy Volunteers. *Nutrients*. 14(19):4094. doi: 10.3390/nu14194094.

REVIEW ON EFFECT OF CLIMATE CHANGE ON COASTAL ECOSYSTEM

Prathamesh Jagdeo Ade*[1], Pratiksha Venudasji Nimbarte[2] and Siddhesh Bhiwa Adkar[3]

[1]Department of Aquatic Environment Management, College of Fisheries, Ratnagiri, M.S.

[2]Department of Aquaculture, College of Fisheries, Ratnagiri, M.S.

[3]Department of Fisheries Biology, College of Fisheries, Ratnagiri, M.S.

*Corresponding author E-mail: praveshade7@gmail.com

Abstract:

Coastal habitats are significantly impacted by climate change, which influences populations and the environment. Coastal landscapes are changing, and these places' resilience is being threatened by factors such as rising sea levels, stronger storms, and altered weather patterns. Seagrass meadows, Salt marshes, and Mangrove forests are the most important part of Coastal ecosystem, which are important for storing atmospheric carbon dioxide, helps in preventing erosion, purify water and offers habitat for variety of species. However, these ecosystems are vulnerable to threats posed by climate change, such as storm surges, increasing sea levels, and human activities like development and pollution. In addition to having an impact on biodiversity, the loss of coastal ecosystems puts coastal populations at higher risk of extreme weather occurrences. Maintaining the health of the environment and civilization, as well as preserving and safeguarding these habitats is necessary to lessen the impact of climate change on coastal areas.

Keywords: Climate Change, Coastal Ecosystem, Seagrasses, Conservation.

Introduction:

Climate change effects on coastal ecosystems are a serious environmental issue that has broad effects on human populations and the environment. Coastal regions are particularly vulnerable to the negative effects of climate change, which include rising sea levels, extreme weather, and altered patterns of precipitation and temperature. The delicate balance of coastal ecosystems, which offer vital functions like carbon sequestration, habitat for biodiversity, and protection against erosion and storm surges, is seriously threatened by these changes. Protecting coastal populations from natural catastrophes like tropical cyclones requires valuing estuarine and coastal ecosystem services above everything else (Barbier *et al.*, 2011). Millions of people's lives are yearly put in danger owing to the destruction of coastal ecosystems brought on by

climate change (Hülsen *et al.,* 2023). The major cause of the loss of natural protection for coastal populations has been shown to be the deterioration of coastal ecosystems, and future forecasts suggest that dangers associated with climate change may affect an extra 27 million people annually.

Climate change long-term implications on coastal and transitional ecosystems have been the focus of numerous studies. The findings have revealed alarming trends in temperature swings, variations in precipitation, and sea level rise. Furthermore, there is a complicated relationship between the health of coastal ecosystems and climate change, with particular attention to the burial rates and temporal records of organic carbon stocks in Mexican blue carbon coastal ecosystems (Cuellar-Martínez *et al.,* 2020). Moreover, a thorough evaluation of the consequences on marine coastal habitats due to climate change has identified the major stressors affecting these ecosystems (Trégarot *et al.,* 2023).

As climate change becomes worse, protecting and restoring coastal habitats becomes more and more important. Investing in the wellbeing of ecological systems like reefs and mangroves is one promising way to mitigate the consequences of climate change on coastal regions. Such solutions have their roots in the natural world. It is feasible to improve coastal communities' resilience and lessen the severity of hazards associated with climate change by preserving and restoring coastal ecosystems. Thus, there are ramifications for both ecological integrity and human well-being from the extensive and varied consequences of climate change on coastal ecosystems. Thus, creating efficient conservation and management plans requires an understanding of how climate change affects coastal ecosystems.

Major impacts on coastal ecosystems

1. Mangrove forests:

Globally, mangrove forests are seriously threatened by climate change, which will affect their resilience, biodiversity, and structure. Mangroves are being impacted at regional scales by interrelated climate change causes, with distinct effects in different geographic regions. These elements include increasing sea levels, increased storminess, changed precipitation regimes, and increasing temperatures. It has been demonstrated via research that mangrove ecosystems are especially susceptible to fluctuations in the duration of inundations, which is one of the primary reasons why sea level rise is such a big problem (Ward *et al.,* 2016). It is anticipated that certain regions, such as North and Central America, Australia, Asia, and East Africa, would be more significantly affected by shifts in storm frequency and severity than South America and West Africa (Ward *et al.,* 2016). Furthermore, mangroves are affected by climate change in ways that go beyond environmental concerns. Globally, mangrove habitats are seriously threatened locally

by human activities including urbanization, agricultural development, and industrialization. Due to nutrient excess from human activity, eutrophication is recognized as a serious danger to mangroves and related ecosystems like coral reefs.

The detrimental effects of human activity on these essential coastal ecosystems are further exacerbated by the proximity of human populations to mangrove regions. Bardou *et al.* (2023) highlight how important it is to comprehend how mangrove ecosystems are impacted by both human activity and climate change. According to their research, mangroves are invading salt marshes because of climate change-related temperature increases, which is changing the mangroves' natural distribution patterns. Recent years have seen a rise in conservation initiatives to preserve and restore mangrove habitats due to their significance. Because of this, the effects of climate change on the mangrove ecosystems are complicated and difficult to predict, affecting not just the ecological integrity of these areas but also the lives of populations dependent on these coastal ecosystems. In order to protect mangrove ecosystems' resilience and long-term viability in the face of growing environmental problems, customized conservation efforts need to take into account regional differences in the risks and effects of climate change.

2. Coral reefs:

Millions of citizens worldwide rely on coral reefs for vital ecosystem services because they are extremely productive and diversified marine ecosystems. However, the effects of climate change are posing hitherto unheard-of risks to these ecosystems. One of the most significant consequences that climate change has on reef systems is a phenomenon known as coral bleaching. Coral bleaching is the process by which corals turn white by expelling the algae that are residing in their tissues. Stressors like high sea temperatures, which are rising due to climate change, start this process (Hoegh-Guldberg *et al.,* 2017). Events of coral bleaching are occurring more frequently and with greater severity due to the rise in water temperatures linked to climate change. For instance, coral reefs in all major reef zones, such as the Great Barrier Reef (Australia), the Pacific and the Caribbean were impacted by the widespread coral bleaching event that happened in 2016 and 2017 (Waycott *et al.,* 2018). These bleaching episodes have resulted in the loss of a great number of corals; in certain reefs, the coral biomass has been reduced by as much as ninety percent (Waycott *et al.,* 2018).

Ocean acidification, or the process by which the ocean takes up atmospheric carbon dioxide and lowers its pH, is another effect of climate change in addition to coral bleaching. Corals are more susceptible to erosion and breaking as a result of ocean acidification, which also makes it more difficult for them to form calcium carbonate-based skeletons (Gattuso *et al.,* 2015). Given that corals provide a vital home for a variety of marine organisms, this might have

detrimental effects on coral reef ecosystems. The corals themselves are not the only thing affected by climate change on coral reefs. A wide variety of marine species, including as invertebrates, fish, and algae, are supported by coral reefs and rely on them for food, shelter, and habitats for reproduction. It is therefore possible that the destruction of coral reefs as a consequence of climate change will have an adverse impact on the marine environment, leading to a decline in the function of ecosystems and the richness of the marine environment (Bellwood *et al.,* 2019). As a result, rising sea temperatures and acidity levels in the ocean are having a major and detrimental impact on coral reefs, leading to an increase in both the severity and frequency of coral bleaching episodes. Widespread coral death is being brought on by these bleaching events, which has detrimental effects on the biodiversity and ecological services that coral reef ecosystems offer.

3. Seagrass beds

Seagrass beds are important coastal ecosystems that offer many marine species' habitats, nitrogen cycling, and sediment stabilization, among other ecological functions. On the other hand, the global wellness and adaptability of seagrass ecosystems are becoming more and more threatened by climate change. One of the primary consequences of climate change on seagrass meadows is sea level rise. When ice caps and glaciers melt as a result of rising global temperatures, sea levels rise. This sea level rise might result in the loss or submersion of seagrass ecosystems (Waycott *et al.,* 2009). One of the main consequences of climate change on seagrass meadows is changes in temperature. Because seagrasses are delicate to temperature fluctuations, even little rises in water temperature can cause stress and even death. Seagrass development rates dramatically decreased as temperatures rose (Marba and Duarte, 2010). Since seagrass beds are a vital source of habitat for a variety of aquatic organisms, including fish, crabs, and mollusks, this might have a domino effect on the environment.

Because of its effects on ocean acidification, climate change can potentially have an influence on seagrass habitats. The seas absorb more carbon dioxide (CO2) as the amount of CO2 in the atmosphere rises, which lowers pH. Due to this, seagrasses may have difficulty absorbing nutrients, which might result in slower development and lower production. In addition, ocean acidification may weaken seagrass blades, making them more susceptible to physical pressures like currents and waves. Seagrass beds are essential to coastal communities' food security and way of life, therefore their disappearance due to climate change might have detrimental effects. Because seagrass beds give fish as well as other commercially significant species a place to live, they sustain profitable fisheries. By retaining nutrients and sediments,

they also assist in preserving the quality of the water, preventing coastal erosion, and safeguarding coral reefs (Orth *et al.,* 2006).

4. Kelp forests:

Kelp forests, characterized by the prevalence of large brown algae, are complex marine ecological systems that serve as crucial habitats and sources of sustenance for a diverse array of marine organisms. Globally, forests of kelp are being significantly impacted by climate change in terms of their structure, biodiversity, and function. The increase in sea temperature is a prominent consequence of climate change that significantly impacts kelp forests. Kelp forests are experiencing strain and are diminishing due to escalating global temperatures, particularly in regions where temperatures exceed their tolerance levels (Wernberg *et al.,* 2016). The distribution and quantity of kelp species can change in response to variations in sea temperature; warmer waters tend to favor the growth of opportunistic, faster-growing species above a slower-growing, canopy-forming species (Krumhansl *et al.,* 2016). This may have an impact on the biodiversity and ecological services that kelp forests offer by altering their shape and function.

Extreme weather events have the potential to completely destroy kelp forests in certain situations, which would have a lasting impact on marine ecosystems ecosystem as well as biodiversity. Because it causes ocean acidification, climate change can potentially have an influence on kelp forests. The seas absorb more carbon dioxide (CO2) as the amount of CO2 in the atmosphere rises, which lowers pH. Due to this, kelp may be less able to absorb minerals and nutrients from the water, which might result in slower growth and production (Krumhansl *et al.,* 2016). Moreover, the susceptibility of kelp plants to physical stressors such as waves and currents may be heightened due to the phenomenon of ocean acidification. Climate change-related kelp forest destruction may have profound ecological and societal effects. For many marine animals, such as fish, insects, and seabirds, kelp forests offer vital habitat. By lowering wave energy and erosion, they also significantly contribute to coastal protection (Reed *et al.,* 2016). Furthermore, kelp forests sustain important tourist and fishing sectors, which give millions of people worldwide access to food and a living. Thus, the resilience and health of kelp forests across the world are facing serious difficulties due to climate change. These significant marine ecosystems are under decline due to a number of factors, including ocean acidification, harsh weather, and rising sea temperatures. To lessen the effects of climate change and save kelp forests for future generations, immediate action is required.

5. Salt marshes:

Salt marshes are highly productive coastal environments that play a vital role in biological processes such as nitrogen cycling, maintaining shoreline stability, and providing

habitat for a diverse range of species. Nevertheless, the worldwide impact of climate change poses a significant danger to the resilience and general health of salt marshes. The rise in sea level is a prominent consequence of climate change that significantly impacts salt marshes. The rise in sea levels may be attributed to the melting of ice caps and glaciers, which is a consequence of the escalating global temperatures.

According to Kirwan *et al.* (2016), the rise in sea level can lead to the erosion and submergence of salt marsh ecosystems. Variations in sea levels can lead to changes in both the distribution as well as the structure of salt marsh ecosystems. According to Kirwan *et al.* (2016), increased sea levels, for instance, may cause the migration of salt marsh habitats inland and the extinction of low-lying marsh regions. This might have a significant effect on the biodiversity and ecological services that salt marshes provide, as well as the communities that rely on these environments for food and livelihoods. Variations in patterns of precipitation and temperature are two other important ways that climate change affects salt marshes. Changes in these climatic factors can have an impact on salt marshes' resilience and output. Because of its effects on severe weather like storms and droughts, climate change can potentially have an impact on salt marsh ecosystems.

Salt marsh ecosystems may sustain physical harm from these occurrences, such as erosion and plant loss (Temmerman *et al.,* 2013). As a result, there may be habitat deterioration and a decrease in resistance to upcoming environmental stresses. There may be substantial ecological and economic repercussions from the loss of salt marshes brought on by climate change. Fish, birds, and invertebrates are just a few of the many species that depend on salt marshes for their home. By lessening the effects of waves and storm surges, they also significantly contribute to coastal protection (Temmerman *et al.,* 2013). Furthermore, salt marshes sustain important fisheries and offer millions of citizens worldwide leisure possibilities. In conclusion, the resilience and health of salt marshes across the world are facing serious difficulties due to climate change. The collapse of these significant coastal ecosystems is attributed to a number of factors, including changes in precipitation and temperature patterns, sea level rise, and extreme weather events. To lessen the effects of climate change and save the salt marshes for future generations, immediate action is required.

6. Biodiversity:

Rising temperatures, an increase in sea level, and ocean acidity all have an influence on coastal biodiversity by changing habitats and endangering species. The distribution and quantity of species are impacted by these changes, which upset ecosystems. Coastal ecosystems that are

particularly vulnerable to deterioration and loss include mangroves, seagrass beds, and coral reefs (Santojanni *et al.*, 2023).

a) Diversity in Threatened Species: Numerous creatures inhabiting coastal environments are susceptible to alterations in both temperature and surroundings. Their survival might be threatened by climate change, which would also significantly affect the biodiversity of the area.

b) Shifts in Species Distribution: Some species may alter their geographic range in response to climate change. Certain species might have to relocate or change their habitats in quest of better ones, upsetting the delicate balance of nearby ecosystems.

c) Loss of Important Ecosystems: Rising sea levels and temperatures can damage and eliminate important ecosystems including seagrass beds, coral reefs, and mangrove forests.

d) Impact on Ecological Balance: Climate change can disturb the balance of coastal ecologies by changing organisms that are part of the food chain. As a result, ecosystems may accidentally change in terms of structure and function.

e) Loss in Ecosystem Services: Coastal habitats provide vital ecosystem services, such as storm blockage, water filtration, and carbon sequestration. Changes to this environment may make it more difficult to provide these services.

f) Storm and Extreme Weather Vulnerability: Climate change may cause storms and other extreme weather events to occur more frequently and with greater intensity in coastal areas. This might cause significant physical harm to ecosystems and habitats.

g) Effect on Human Resources: For many human groups, coastal ecosystems are vital resources. The tourism industry, coastal communities overall, and fishermen's livelihoods may all be at risk due to climate change that negatively impacts this environment.

h) Threat of Species Loss: If climate change dramatically disrupts the habitats of endemic species, which are exclusively found in coastal locations, then such species may be in danger of becoming extinct. This can be a significant drawback to the preservation of biodiversity.

Management strategies:

The primary objectives of mitigation include lowering greenhouse gas emissions, converting to renewable energy sources, boosting energy efficiency, sustainable agriculture, waste management, implementing carbon pricing mechanisms, afforestation, promoting alternative forms of transportation, and streamlining industrial processes. These initiatives seek to mitigate the effects of global warming, which are essential for protecting species and coastal ecosystems. However, adaptation entails making adjustments to address the consequences of

climate change. Creating infrastructure that is climate resilient, adjusting ecosystems, managing water resources, adapting agriculture, developing early warning systems, enhancing healthcare, including the community, and managing risks and insurance are some strategies. By strengthening resilience and lowering vulnerability, these actions support ecosystems' and societies' adaptation to climate change.

Important adaptation measures are required to solve the problems that climate change is posing to biodiversity and coastal environments. A few of these include biological conservation, ecosystem monitoring, community-based adaptation, resource-based adaptation, disaster risk reduction, resource-based adaptation, careful management of water and marine resources, biological conservation, the use of marine protected areas, resource-based adaptation, and raising awareness. By putting these techniques into practice, human populations and coastal ecosystems can adapt to climate change more effectively, preserving important species and advancing long-term sustainability and conservation objectives. To effectively handle the issues posed by climate change, cooperation between nations and global organizations is essential, particularly when it comes to changes in the movement and distribution of species. In order to engage the public and promote action to reduce and adapt to the effects of climate change, education, and awareness are essential for securing the future sustainability of coastal ecosystems and biodiversity.

Conclusion:

This review looked closely at how climate change affects coastal biodiversity, emphasizing how urgently mitigation and adaptation plans are needed. The assessment examined the serious risks that ocean acidification, sea level rise, and warming temperatures pose to species and coastal environments. It emphasized how crucial it is to conduct coordinated conservation initiatives in order to save delicate ecosystems including coral reefs, mangroves, and seagrass beds. The mitigation techniques that were deliberated encompassed switching to sustainable energy sources, enhancing energy efficiency, enacting carbon pricing systems, planting trees, practicing sustainable agriculture, handling garbage, and endorsing other modes of transportation. In order to protect coastal biodiversity, it is imperative that greenhouse gas emissions be reduced and the rate of global warming be limited. Building climate-resilient infrastructure, ecosystem-based adaptation, water management, agricultural adaptation, early warning systems, healthcare advancements, community participation, insurance, and risk management were among the equally important adaptation techniques that were described. In order to minimize vulnerability, build resilience, and provide assistance to ecosystems and communities in adapting to the consequences of climate change, these strategies are being implemented.

The review also stressed upon significance of certain adaption tactics, such as genetic conservation, protected marine areas, environmentally friendly resource management, habitat protection, and ecosystem restoration, for coastal ecosystems and biodiversity. These tactics can support long-term coastal biodiversity conservation and sustainability, in addition to international cooperation and educational initiatives. All things considered, this analysis offers a thorough summary of the threats that climate change poses to coastal biodiversity and emphasizes the pressing need for action. The implementation of adaptation and mitigation strategies is the means by which we may decrease the impact of climate change, conserve coastal ecosystems, and provide a sustainable future for the biodiversity of coastal areas.

References:

1. Barbier, E.B. *et al.* (2011). The value of estuarine and coastal ecosystem services. Ecological Monographs, 81, 169–193.
2. Bardou, Rémi & Osland, Michael & Scyphers, Steven & Shepard, Christine & Aerni, Karen & Alemu I, Jahson & Crimian, Robert & Day, Richard & Enwright, Nicholas & Feher, Laura & Gibbs, Sarah & O'Donnell, Kiera & Swinea, Savannah & Thorne, Kalaina & Truskey, Sarit & Armitage, Anna & Baker, Ronald & Breithaupt, Joshua & Cavanaugh, Kyle & Hughes, Anne. (2023). Rapidly Changing Range Limits in a Warming World: Critical Data Limitations and Knowledge Gaps for Advancing Understanding of Mangrove Range Dynamics in the Southeastern USA. Estuaries and Coasts. 46. 10.1007/s12237-023-01209-7.
3. Bellwood, David & Pratchett, Morgan & Morrison, Tiffany & Gurney, Georgina & Hughes, Terence & Álvarez-Romero, Jorge & Day, Jon & Grantham, Ruby & Grech, Alana & Hoey, Andrew & Jones, Geoffrey & Pandolfi, J. & Tebbett, Sterling & Techera, Erika & Weeks, Rebecca & Cumming, Graeme. (2019). Coral reef conservation in the Anthropocene: Confronting spatial mismatches and prioritizing functions. Biological Conservation. 236. 10.1016/j.biocon.2019.05.056.
4. Cuellar-Martínez, T. *et al.* (2020). Temporal records of organic carbon stocks and burial rates in Mexican blue carbon coastal ecosystems throughout the globe. Planet. Chang. Duarte, Carlos & Marba, Nuria & Gacia, Esperança & Fourqurean, James & Beggins, Jeff & Barrón, Cristina & Apostolaki, Eugenia. (2010). Seagrass community metabolism: Assessing the carbon sink capacity of seagrass meadows. Global Biogeochemical Cycles. 24. 10.1029/2010GB003793.
5. Gattuso, Jean-Pierre & Magnan, Alexandre & Billé, Raphaël & Cheung, William & Howes, Ella & Joos, Fortunat & Allemand, Denis & Bopp, Laurent & Cooley, Sarah &

Eakin, C. Mark & Hoegh-Guldberg, Ove & Kelly, Ryan & Pörtner, Hans-Otto & Rogers, Alex & Baxter, John & Laffoley, Dan & Osborn, D & Rankovic, A & Rochette, J & Turley, Carol. (2015). Contrasting futures for ocean and society from different anthropogenic CO_2 emissions scenarios. Science. 349. aac4722. 10.1126/science.aac4722.

6. Hoegh-Guldberg, Ove & Poloczanska, Elvira & Skirving, William & Dove, Sophie. (2017). Coral Reef Ecosystems under Climate Change and Ocean Acidification. Frontiers in Marine Science. 4. 10.3389/fmars.2017.00158.
7. Hughes, Terence & Kerry, James & Alvarez-Noriega, Mariana & Álvarez-Romero, Jorge & King, Kristen & Baird, Andrew & Babcock, R. & Beger, Maria & Bellwood, David & Berkelmans, Ray & Bridge, Tom & Butler, Ian & Byrne, Maria & Cantin, Neal & Comeau, Steeve & Connolly, Sean & Cumming, Graeme & Dalton, Steven & Diaz-Pulido, Guillermo & Wilson, Shaun. (2017). Global warming and recurrent mass bleaching of corals. Nature. 543. 373-377. 10.1038/nature21707.
8. Hülsen, S. *et al.* (2023). Global protection from tropical cyclones by coastal ecosystems – past, present, and under climate change. Environmental Research Letters, 18, 124023.
9. Kirwan, Matthew & Walters, David & Reay, William & Carr, Joel. (2016). Sea level driven marsh expansion in a coupled model of marsh erosion and migration: Sea Level Driven Marsh Expansion. Geophysical Research Letters. 43. 10.1002/2016GL068507.
10. Krumhansl, Kira & Okamoto, Daniel & Rassweiler, Andrew & Novak, Mark & Bolton, J. & Cavanaugh, Kyle & Connell, Sean & Johnson, Craig & Konar, Brenda & Ling, Scott & Micheli, Fiorenza & Norderhaug, Kjell & Perez-Matus, Alejandro & Sousa Pinto, Isabel & Reed, Daniel & Salomon, Anne & Shears, Nick & Wernberg, Thomas & Anderson, Robert & Byrnes, Jarrett. (2016). Global patterns of kelp forest change over the past half-century. Proceedings of the National Academy of Sciences of the United States of America. 113. 10.1073/pnas.1606102113.
11. Marba, Nuria & Duarte, Carlos. (2010). Mediterranean warming triggers seagrass (Posidonia oceanica) shoot mortality. Global Change Biology. 16. 2366 - 2375. 10.1111/j.1365-2486.2009.02130.x.
12. Orth, Robert & Carruthers, Tim & Dennison, William & Duarte, Carlos & Fourqurean, James & JR, KENNETH & Hughes, Anne & Kendrick, Gary & Kenworthy, W. & Olyarnik, Suzanne & Short, Frederick & Waycott, Michelle & Williams, Susan. (2006). A Global Crisis for Seagrass Ecosystems. BioScience. 56. 987-996. 10.1641/0006-3568(2006)56[987: AGCFSE]2.0.CO;2.

13. Reed, Daniel & Washburn, Libe & Rassweiler, Andrew & Miller, Robert & Bell, Tom & Harrer, Shannon. (2016). Extreme warming challenges the sentinel status of kelp forests as indicators of climate change. Nature Communications. 7. 10.1038/ncomms13757.
14. Santojanni, Fourqurean & Miner, Howe & Hain, Hain & Sutton, Graham. (2023). The Impact of Climate Change on Biodiversity in Coastal Ecosystems. Jurnal Ilmu Pendidikan dan Humaniora. 12. 167-182. 10.35335/jiph.v12i3.9.
15. Temmerman, Stijn & Meire, Patrick & Bouma, Tjeerd & Herman, Peter & Ysebaert, Tom & de Vriend, Huib. (2013). Ecosystem-based coastal defense in the face of global change. Nature. 504. 79-83. 10.1038/nature12859.
16. Trégarot, Ewan & D'Olivo, Juan & Botelho, Andrea & Cabrito, Andrea & Cardoso, Gabriel & Casal, Gema & Cornet, Cindy & Cragg, Simon & Degia, A. Karima & Fredriksen, Stein & Furlan, Elisa & Heiss, Georg & Kersting, Diego & Maréchal, Jean-Philippe & Meesters, Erik & O'Leary, Bethan & Pérez, Géraldine & Seijo-Núñez, Cristina & Simide, Remy & Juan, Silvia. (2023). Effects of climate change on marine coastal ecosystems -A review to guide research and management. Biological Conservation. 289. 110394. 10.1016/j.biocon.2023.110394.
17. Ward, Raymond & Friess, Dan & Day, Richard & Mackenzie, Richard. (2016). Impacts of climate change on mangrove ecosystems: a region by region overview. Ecosystem Health and Sustainability. 2. 1-25. 10.1002/ehs2.1211.
18. Waycott, Michelle & Duarte, Carlos & Carruthers, Tim & Orth, Robert & Dennison, William & Olyarnik, Suzanne & Calladine, Ainsley & Fourqurean, James & Heck, Ken & Hughes, Anne & Kendrick, Gary & Kenworthy, W. & Short, Frederick & Williams, Susan. (2009). Accelerating loss of seagrass across the globe threatens coastal ecosystems. Proceedings of the National Academy of Sciences of the United States of America. 106. 12377-81. 10.1073/pnas.0905620106.
19. Wernberg, Thomas & Bennett, Scott & Babcock, R. & Bettignies, Thibaut & Cure, Katherine & Depczynski, Martial & Dufois, Francois & Fromont, Jane & Fulton, Christopher & Hovey, Renae & Harvey, Euan & Holmes, Thomas & Kendrick, Gary & Radford, Ben & Santana-Garcon, Julia & Saunders, Benjamin & Smale, Dan & Thomsen, Mads & Tuckett, Chenae & Wilson, Shaun. (2016). Climate-driven regime shift of a temperate marine ecosystem. Science. 353. 169-172.

IMPACT OF COSMETICS PRODUCTS ON ENVIRONMENT

N. P. Sanap

Department of Zoology,

Shardchandra Arts, Commerce and Science College, Naigaon. Dist. Nanded.

Corresponding author E-mail: npsanap@gmail.com

Abstract:

Humans have studied numerous environmental pollution issues and improved treatment in the past, but few people are aware that cosmetics are also potential environmental pollution factors. This article first discusses that most cosmetics contain hazardous substances and then examines the effects of these hazardous substances on the natural environment, organisms, and humans. According to studies, cosmetics primarily contain heavy metals, organics and other hazardous substances which can pollute the environment's water and soil, organism reproduction and growth and cause a variety of physiological ailments in humans. The article then proposes two technologies for effectively treating hazardous substances in cosmetics: biosorbent and activated carbon fiber-polyethersulfone (ACF-PES) ultra filtration composite membrane, both of which adsorbs and effectively degrade hazardous substances via their respective physical and chemical properties. Moreover, this article examines the necessity and feasibility of measures for the government, corporations and the general public to participate in the treatment of hazardous substances in cosmetics. Legislation to regulate and supervise cosmetics production should be strengthened by the government. Corporations should manufacture green cosmetics and promote the use of green cosmetics. To limit the impact of hazardous substances in cosmetics, the general public can employ strategies such as using less or purchasing cosmetics containing less dangerous substances. The article promotes a greater understanding of the dangers of harmful substances in cosmetics and offers some suggestions for reducing their effects.

Keywords: Cosmetics, Human Use, Environment, Adverse Effects.

Introduction:

Cosmetics have become an essential aspect of people's lives in recent times. In the previous three decades, the market demand for cosmetics has always been on the increase [1]. While cosmetics enrich our quality of life, some inescapable issues have arisen. The majority of cosmetic items are over packaged. In the preservation and packaging of cosmetics, a great number of paper goods, plastics, and glass are utilized, resulting in a large amount of waste. This

issue has been identified by scientists and the government. Scientists have conducted extensive research on cosmetic plastics, and some governments have restricted or prohibited the use of plastic goods to package cosmetics in order to address environmental concerns. Many people, however, overlook the cosmetics themselves. Cosmetics are human beauticians, but they are also potential environmental and organism killers. Some cosmetics ingredients used to enhance the effect may be dangerous. However, if these hazardous substances enter nature and exceed the carrying capacity of organisms and the environment, they will have an impact not just on humans but also on the entire ecosystem. Because of hazardous substances in cosmetics, the system and the entire global ecosystem may be severely harmed and difficult to restore. There are many cosmetics-related articles, such as those analyzing a specific hazardous ingredient in cosmetics, the influence of cosmetic hazardous substances in a specific area, and the harm caused by cosmetic hazardous substances to a specific organism. However, these articles frequently investigate only one type of cosmetic or one type of cosmetic hazardous ingredient and there are few complete evaluations. Furthermore, most articles only analyze the types of hazardous substances in cosmetics and the harm they cause, rarely providing relevant technologies for scientific treatment, and ignoring the fact that the government, corporations, and the general public can all play an important role in dealing with hazardous substances in cosmetics. This article discusses the hazardous substances found in cosmetics and the effects they have on the environment, organisms and humans. Two technologies for efficiently dealing with hazardous substances in cosmetics are advocated, as well as the participation of the government, corporations and the general public. The article aims to raise awareness of the public for the dangers of hazardous substances in cosmetics as well as what possible technologies and ways exist to improve or solve the problem.

Hazardous substances in cosmetics

1. Heavy metals:

Mercury, lead, cadmium, chromium, arsenic, cobalt, and nickel are common heavy metals in cosmetics and there are generally heavy metals in cosmetics such as lipstick, blush, foundation, eye shadow and so on. Heavy metals are not intentionally added to cosmetics, yet many talc and pigments used in cosmetics contain heavy metals, making heavy metals in cosmetics unavoidable. The most prevalent is whitening cosmetics, which frequently include excessive amounts of heavy metals, particularly mercury, which is thousands or even tens of thousands of times greater than safety standards.

2. Organics hormones:

These are one of the organics added to cosmetics that are primarily used for the skin and are generally steroid hormones. Steroid hormones can be split into two categories based on pharmacological analysis: sex hormones and adrenal corticoids. Because glucocorticoids are the most commonly utilized form of steroid hormone, they are the primary detection object in the cosmetics detection procedure and the common hydrocortisone and dexamethasone in cosmetics are all glucose metabolic corticoids

3. Other organic chemicals:

Several other organic chemicals are added to cosmetics to boost their effects. To begin with, phthalates are widely used in daily necessities. They serve a number of purposes, the most important of which is as a solubilizer. Phthalates, for example, are utilized in shampoos to stabilize the substance in liquid form Phthalates are humectants that are used in cosmetics to improve the moisturizing and hydrating properties of emollient products. Secondly, because of their low cost, parabens are used as preservatives and antibacterial agents in cosmetics, and they are stable at different temperatures and have a good sterilizing action Furthermore, triclosan is a common bacteria inhibitor found in soaps and cosmetic cleansers.

4. Cosmetic microorganisms

Others with the exception of heavy metals and organics, some cosmetic microorganisms exceed minimum standards and contain pathogenic microbes that cause significant harm to life. Some cosmetics, for example, include *Staphylococcus aurous*, *Pseudomonas aeruginosa*, *Escherichia coli*, and *Bacillus*.

The emergence of microbes can be attributed to a variety of factors. Cosmetic raw materials are not thoroughly disinfected, cosmetic manufacturing equipment is not cleaned and disinfected regularly, and personnel do not adhere to hygiene standards. Cosmetics may be contaminated by microorganisms found in hair, perspiration or hands. At the same time, cosmetics are good areas for microorganisms to live. Cosmetics are often neutral or mildly acid-alkaline, and they are typically stored at ambient temperature to provide an external environment for microorganisms. Cosmetics include a lot of water and a lot of nutrients for microorganism growth. Furthermore, there is a class of minerals, such as mica, talc, and quartz, that is ground into powder and used in cosmetics such as compacts or sprays and which typically have a function in increasing skin gloss. These minerals are made up of numerous elemental compounds that may undergo chemical interactions throughout the cosmetics manufacturing process, resulting in higher harm.

The effect of hazardous substances

1. Environment

Water pollution Hazardous substances enter the water body in essentially the following ways. The first is that cosmetics are dumped directly into the oceans, rivers and lakes along with sewage. The second type of circumstance is more common by the coast or in densely populated places. Cosmetics containers are dumped in the oceans or rivers, releasing hazardous substances from the cosmetics into the water body. Water pollution is classified as surface water pollution and groundwater pollution and the two are interconnected 2. Surface water will permeate the earth and create groundwater and groundwater can also be delivered upwards to the surface water source. As a result, when hazardous substances spill, they pollute both the surface and groundwater. When hazardous substances enter the water, their concentration gradually decreases at a very slow rate due to water's self-regulation ability, but the rate of concentration degradation is not even faster than the rate of discharge. So, whether heavy metals or hormones are dumped into water bodies, they will have irreversible impacts. For rivers and oceans, continuously accumulating hazardous substances in rivers and oceans will spread from a single area to the world's oceans along with currents or ocean currents; for lakes, because their self-regulation ability is far less than that of rivers and oceans, hazardous substances continuously entering the lake will lead to the disintegration of the lake's ecological environment; for groundwater, although groundwater is less likely to be contaminated by hazardous substances than other bodies of water, it is frequently utilized for animal or human drinking and agricultural irrigation, which can easily injure humans, animals and plants.

2. Soil pollution

The poisoning of the soil with hazardous cosmetic substances is mostly caused by the penetration of cosmetics into the soil combined with sewage or solid waste. These hazardous substances will be absorbed due to the presence of pores in the soil. Hazardous substances, such as heavy metals and hormones, might react with the soil's natural components and kill microorganisms. These are the most essential factors influencing soil fertility and activity. Plants grown in this type of soil will absorb nutrients through their roots while also inhaling a significant number of hazardous substances, which may affect advanced organisms along the food chain and the soil itself may degenerate and change as hazardous substances accumulate. If it is unsuitable for planting, it will result in a slew of issues relating to a lack of available land for agricultural development.

3. Organisms

The way of spreading Animals and plants living in nature will inevitably be harmed if dangerous cosmetics ingredients are released into the environment. Heavy metals and hormones are mostly absorbed into the bodies of animals through two routes: foraging or direct touch and they are continuously deposited in the animals' liver and kidneys. In the process of absorbing water and nutrients from the soil, plant roots absorb hazardous substances in the soil by ion exchange and hazardous substances are delivered to each section of the plant via the plant's channel organization. The process of bioaccumulation is the accumulation of dangerous substances in the body of a single organism. Bioaccumulation occurs when an organism absorbs hazardous substances quicker than the rate of catabolism and excretion to eliminate hazardous substances and toxic substances are frequently digested extremely slowly in the organism, causing them to accumulate Hazardous substances will enter the food chain and be biomagnified, affecting the entire biosphere. The term "biomagnification" refers to the continuous accumulation of hazardous substances in the food chain, i.e., the accumulation of hazardous substances from the lowest producer to the most evolved predator.

The harm on organisms' Hazardous substances in cosmetics have a significant impact on the organism. First, consider heavy metals. According to the research of Zwolak *et al.* and Wang *et al.,* the biological risks of different heavy metals can be arranged in the following order: Hg>Cu>Zn>Ni>Pb>Cd>Cr>Sn>Fe>Mn>Al [10-11]. Take the fish as an example. Fish growth can be hindered in heavy metal-rich water, making growth inhibition one of the most visible indicators of metal toxicity in fish larvae, Water bodies with low heavy metal concentrations will not directly kill the fish but will create chronic stress, resulting in a drop in fish weight and size. Heavy metals can affect juvenile fish survival and growth, as well as produce atypical behaviors such as impaired athletic ability or structural damage, particularly spinal malformations. As a result, their ability to compete for food and habitat is reduced, perhaps leading to fish death or extinction. Then there are hormones. As an example, consider estrogen (E2). It has been determined that estrogen residues can affect the animal endocrine activity and impair sexual performance. Among them, E2 residue is a type of endocrine disruptor with significant influence, with a particularly deleterious effect on fish [14-16]. E2 can accumulate in fish bile, ovaries, and testes, destroying natural fish activities. Male fish in rivers can produce feminine features, such as Vitellogenin induction and gonadal alterations, when estrogen is present.

4. Humans

Humans are also at risk from hazardous substances included in cosmetics. The first category is heavy metals. Humans exposed to high amounts of cadmium (Cd) in a short period of

time may have a variety of health problems such as diarrhea, vomiting, fever, lung damage, muscle soreness, and so on . If you eat cadmium over an extended period of time, it can lead to more serious ailments such as kidney disease and bone damage, reproductive issues, and even cancer. Chromium (Cr) is essential in the body's metabolic process, regulating blood sugar levels and assisting insulin in delivering glucose to cells. However, only a very little quantity of chromium is required, and surpassing a specific threshold, particularly hexavalent chromium, is associated with a significant injury to the human body. Chromium is not hazardous in and of itself, but its compounds are poisonous and can cause widespread disease in the kidneys, liver, nervous system, and blood, ultimately leading to death. Lead (Pb) is one of the most common heavy metals found in cosmetics and it can directly harm the body's major organs and systems. Lead-related disorders include kidney failure, hematological system diseases, cardiovascular diseases, nervous system diseases, and immune system impacts. Humans exposed to low amounts of arsenic (As) may experience vomiting, irregular heartbeat, blood vessel damage, abdominal pain, and diarrhea, numbness, and muscle cramps. When humans are exposed to high levels of arsenic for an extended period of time, the symptoms mainly emerge in the skin, including changes in pigmentation, skin damage, and hard areas on the palms, and in extreme circumstances, it can cause lung cancer, skin cancer, bladder cancer, and kidney cancer. Furthermore, heavy metals in cosmetics, such as nickel, can damage immunity, memory, and exercise ability, as well as induce many types of cancer, which can be extremely harmful to people. Furthermore, several organic substances found in cosmetics have varying degrees of effect on the human body. Long-term glucocorticoid therapy has been shown in trials to cause a variety of adverse effects, including "full moon face," obesity, delayed growth and development, metabolic abnormalities, decreased bone density, and anxiety and depression, particularly in younger children. Phthalates have a negative impact on the male reproductive system in animals, causing hypospadias and cryptorchidism, which diminish testosterone production and sperm counts. Phthalates are also endocrine disruptors.

Government, corporation, public participation

Government Relevant government departments, as legislators and corporate regulators, must assure the quality and safety of cosmetics. The first step is to put in place legally binding regulations for the cosmetics industry, such as manufacturing process standards and waste disposal methods, the maximum use of various chemical additives in cosmetics, the highest content of hazardous substances in cosmetics, and some cosmetics-related regulations and laws. As shown in Table 1, certain nations have implemented necessary laws that regulate the cosmetics industry's production standards and strictly limit the content of hazardous substances

in cosmetics. The second task is to monitor the cosmetics sector. Check if cosmetics on the market fulfill regulatory standards at random or whether cosmetic factories release wastewater and hazardous substances at will, remove non-compliant items from the shelves, and penalize enterprises that make non-compliant cosmetics.

Corporation:

Cosmetics companies should follow national rules and regulations, accept oversight from relevant government departments and consumers, and promote the concept of purchasing eco-friendly products and appropriate purchases to consumers. The most essential task of the cosmetic industry is to manufacture green cosmetics. When manufacturing green cosmetics, it is critical to guarantee that the product adheres to green chemistry principles. Green chemistry encompasses the full process of chemical design, manufacture, and application, and adhering to green chemistry principles can effectively decrease or remove hazardous substances and lessen cosmetics' environmental impact [30- 31]. Public: The general public, as customers, should also be involved. The first is to refuse to purchase cosmetics that contain excessive levels of hazardous substances in order to safeguard the environment while also preserving our safety. Natural cosmetics, also known as green cosmetics, are made from extracts and concentrations of plants or fruits. Green cosmetics do not employ chemical substances or other nonnatural mixes in the manufacturing process, and they require less water, raw materials, and energy than conventional cosmetics, resulting in less pollution of the natural environment [33-34]. The second is reasonable garbage disposal cosmetics. As an example, when we go to the beach, we often witness individuals throwing away sunscreen bottles after using them, generating pollution. Cosmetics containers may have residual cosmetics. It is simple to pollute water or soil if they are discarded at will. Choose to dispose of cosmetics in the trash can, and the reusable plastic or paper box will be recycled by the garbage disposal station, which is a more environmentally responsible practice.

Conclusion:

Cosmetics are widely utilized in daily life, and the influence of their hazardous constituents on the overall environment is frequently disregarded. This study shows that cosmetics contain harmful substances such as heavy metals, organic substances, and microorganisms. When they enter the environment, they may cause water and soil pollution, and may also accompany the food chain, causing health effects and even death at all levels of the organism. The article proposed two technologies to alleviate the pollution problem caused by hazardous substances: biosorbents and ACF-PES ultra filtration composite membrane. At the same time, the article examines the governments, corporations', and individuals' responsibilities

and obligations in the face of hazardous substances in cosmetics, as well as proposed solutions to the problems. If more equipment and advanced technology can be applied to the research on hazardous substances in cosmetics in the future, it may be possible to avoid the current research on only a single type of hazardous substance in cosmetics rather than all hazardous substances. This makes determining if there may be secondary reactions between hazardous substances that cause other effects impossible. The purpose of writing this article was to raise general awareness, provide a theoretical foundation to demonstrate the harmfulness of hazardous substances, and offer specific strategies to eliminate them in cosmetics.

References:

1. Alam, M., Akhter, M., Mazumder, B., Ferdous, A., Hossain, M., Dafader, N., Ahmed, F., Kundu, S., Taheri, T., & Ullah, A. A. (2019). Assessment of some heavy metals in selected cosmetics commonly used in Bangladesh and human health risk. *Journal of Analytical Science and Technology, 10*(1), 1-8.
2. Amenyogbe, E., Chen, G., Wang, Z., Lu, X., Lin, M., Lin, A. Y., (2020). A review on sex steroid hormone estrogen receptors in mammals and fish. *International Journal of Endocrinology, 2020*.
3. Gibson, R., Smith, M., Spary, C., Tyler, C., & Hill, E. (2005). Mixtures of estrogenic contaminants in bile of fish exposed to wastewater treatment works effluents. *Environmental Science & Technology*, *39*(8), 2461-2471.
4. Gray, J. S. (2002). Biomagnification in marine systems: The perspective of an ecologist. *Marine Pollution Bulletin, 45*(1-12), 46-52.
5. Guo, Y., & Kannan, K. (2013). A survey of phthalates and parabens in personal care products from the United States and its implications for human exposure. *Environmental Science & Technology, 47*(24), 14442-14449.
6. Karpuzoglu, E., Holladay, S. D., & Gogal Jr, R. M. (2013). Parabens: potential impact of low-affinity estrogen receptor binding chemicals on human health. *Journal of Toxicology and Environmental Health, Part B, 16*(5), 321-335.
7. Khayatzadeh, J., & Abbasi, E. (2010). The effects of heavy metals on aquatic animals. In *The 1st International Applied Geological Congress* (pp. 26-28). Department of Geology, Islamic Azad University–Mashad Branch, Iran.
8. Kim, H. W., Seok, Y. S., Cho, T. J., & Rhee, M. S. (2020). Risk factors influencing contamination of customized cosmetics made on-the-spot: Evidence from the national pilot project for public health. *Scientific Reports, 10*(1), 1-9.

9. Łopaciuk, A., & Łoboda, M. (2013). Global beauty industry trends in the 21st century. In *Management, Knowledge and Learning International Conference* (pp. 19-21).
10. Müller, A.-K., Markert, N., Leser, K., Kämpfer, D., Crawford, S. E., Schäffer, A., Segner, H., & Hollert, H. (2020). Assessing endocrine disruption in freshwater fish species from a "hotspot" for estrogenic activity in sediment. *Environmental Pollution, 257*, 113636.
11. Paulsen, L. (2015). The health risks of chemicals in personal care products and their fate in the environment.
12. Soni, H. B. (2019). Categories, causes and control of water pollution - A review by Hiren B. Soni. *Life Sciences Leaflets, 107*, 4-12.
13. Sun, S.-X., Zhang, Y.-N., Lu, D.-L., Wang, W.-L., Limbu, S. M., Chen, L.-Q., Zhang, M.-L., & Du, Z.-Y. (2019). Concentration-dependent effects of 17β-estradiol and bisphenol A on lipid deposition, inflammation and antioxidant response in male zebrafish (*Danio rerio*). *Chemosphere, 237*, 124422.
14. Uraipong, C., Allan, R. D., Li, C., Kennedy, I. R., Wong, V., & Lee, N. A. (2018). 17β-Estradiol residues and estrogenic activities in the Hawkesbury River, Australia. *Ecotoxicology and Environmental Safety, 164*, 363-369.
15. Wang, Q.-R., Cui, Y.-S., Liu, X.-M., Dong, Y.-T., & Christie, P. (2003). Soil contamination and plant uptake of heavy metals at polluted sites in China. *Journal of Environmental Science and Health, Part A, 38*(5), 823-838.
16. Zhou, X., Yang, Z., Luo, Z., Li, H., & Chen, G. (2019). Endocrine disrupting chemicals in wild freshwater fishes: Species, tissues, sizes and human health risks. *Environmental Pollution, 244*, 462-468.
17. Zwolak, A., Sarzyńska, M., Szpyrka, E., & Stawarczyk, K. (2019). Sources of soil pollution by heavy metals and their accumulation in vegetables: A review. *Water, Air, & Soil Pollution, 230*(7), 1-9.

ENSURING THE FUTURE OF FISHERIES IN ASSAM: NAVIGATING CHALLENGES AND EMBRACING OPPORTUNITIES

Liza Dutta[1], Kollol Pratim Baruah[2] and Gaurav Phookan[2]

[1]Department of Fisheries Science, Alagappa University, Tamil Nadu

[2]Department of Agriculture, Govt. of Assam

*Corresponding author E-mail: lizadutta2002@gmail.com

Abstract:

Situated in the northeastern region of India, Assam boasts abundant aquatic resources, including extensive river systems, floodplain lakes, and ponds, which serve as traditional fishing grounds. Despite its significance, the fisheries sector faces multifaceted challenges that impede its sustainable development. This book chapter explores the intricate dynamics of the fisheries sector in Assam, India, shedding light on its pivotal role in the state's economic landscape. Through an in-depth analysis, this chapter delves into the various challenges encountered by fish farmers in Assam, ranging from production-related issues such as inadequate technical assistance and scarcity of inputs to price-related challenges like irregular payments and marketing-related obstacles. Furthermore, the chapter highlights supplementary challenges, including the lack of government support and technical assistance, which further hinder the sector's growth and development. In addition to examining the challenges, this chapter also identifies the opportunities present in Assam's fisheries sector. With its remarkable growth trajectory and untapped potential, the sector holds promise for economic growth, employment generation, and food security. By addressing the identified challenges and embracing sustainable management practices, Assam can harness the full potential of its fisheries resources, ensuring prosperity for both fish farmers and the broader community. This book chapter aims to provide valuable insights into the complexities of sustaining fisheries in Assam and offers recommendations for policymakers, industry stakeholders, and support organizations to foster the sector's resilience and long-term viability. Through collaborative efforts and targeted interventions, Assam can emerge as a model for sustainable fisheries management, setting a precedent for other regions facing similar challenges worldwide.

Keywords: Fisheries, Challengs, Opportunitoes, Assam

Introduction:

Assam, nestled in the North-East region of India, shares its borders with seven states, Arunachal Pradesh, Manipur, Meghalaya, Mizoram, Nagaland, Tripura, and West Bengal and two neighboring countries, Bangladesh and Bhutan. With a total geographical area of 78,438 sq. km, it comprises 33 districts, including six newly created districts namely Biswanath, Charaideo, Hojai, South Salmara/ Mankachar, West Karbi Anglong, and Majuli. As per the 2011 census, Assam shelters approximately 2.6% of the country's population and encompasses about 2.4% of the nation's total geographical area. Fishery plays a pivotal role in the economic landscape of Assam, contributing significantly to income generation and employment opportunities across the state. Endowed with abundant aquatic resources, Assam's fisheries development is supported by its primary rivers, the Brahmaputra and the Barak, alongside their 53 tributaries, as well as numerous floodplain lakes (beels) and ponds, which constitute traditional fishing grounds.

The undulating topography and ample rainfall further enrich the state's fisheries resources, providing a fertile ground for development. Rice and fish form the fundamental components of the Assamese diet, with a staggering 88.5% of households in Assam including fish in their daily culinary repertoire. Rice cultivation sprawls across nearly 25 lakh hectares of land, with Assam Agricultural University's varieties commanding over 70% of this expanse, as per Dutta *et al.* (2023). Subsistence fishing serves as a cornerstone of the state's economy, especially in rural regions, where it not only supports livelihoods but also bolsters food security. However, the economic importance of subsistence fishing is frequently underestimated due to underreporting, potentially leading to an undervaluation of the fisheries sector. While sectors like tea and rice have been extensively documented, including the tourism facilitated by the tea industry, as highlighted in many of their studies by Dutta *et al.* and Hazarika *et al.*, the crucial role of subsistence fishing warrants greater recognition and acknowledgment. Fish holds substantial importance as a food source globally, accounting for nearly one-fifth of the world's animal protein intake. In India, fishing not only provides sustenance but also livelihoods for millions while contributing valuable foreign exchange earnings. However, the perishable nature of fish necessitates proper handling, processing, and distribution to maintain its quality. Fisheries stand as a crucial sector alongside agriculture, providing employment and ensuring food supply. With approximately 35% of the Indian population being fish eaters, the per capita consumption stands at 9.8 kg, slightly below the recommended intake of 13 kgs.

Fishery sector of Assam:

In India, both marine and inland fisheries play crucial roles in the economy, providing significant contributions. Particularly in states like Assam, inland fisheries serve as essential

sources of employment and sustenance for local fishers. Despite their importance, the fishery sector's contribution to India's Gross Value Added (GVA) was relatively low at 0.96% in 2016-17, compared to its substantial impact on the agriculture sector, which stood at 5.37%. In Assam, the fish production reached 3.31 lakh metric tons in 2018-19, slightly below the estimated nutritional demand of 3.42 lakh metric tons. Additionally, the fishery sector made a notable economic contribution, adding Rs. 7646.71 crores to the Gross State Domestic Product of Assam in the same year.

Assam is blessed with abundant natural resources for fisheries, including ponds, water bodies, and expansive wetlands covering 2.59 lakh hectares. The presence of major river systems like the Brahmaputra, Barak, and their tributaries offers extensive riverine fishery opportunities, spanning 11,304 kilometers. Recognizing this potential, both the central and state governments are prioritizing efforts to bolster the sector, aiming to achieve self-sufficiency in fish production and establish Assam as a prominent hub for fish exports.

Over the years, fish production in Assam has witnessed remarkable growth, escalating from 218 thousand tons in 2009-10 to 4.43 lakh tons in 2022-23. Concurrently, per capita consumption has also seen an increase, rising from 12.18 kg to 13.06 kg. However, despite this progress, scientific fish farming practices are currently utilized in only 5% of the total water resource area, indicating substantial untapped potential for further development and sustainability in the sector.

The changes in water spread areas of various types of fisheries over time in Assam reflect dynamic shifts influenced by a combination of environmental, social, and economic factors. Understanding these changes is essential for effective resource management and sustainable development in the fisheries sector.

1. **River fisheries:** The significant decrease in water spread area from 205,000 hectares in 2009-10 to 11,304.5 hectares in 2022-23 suggests several underlying factors. Environmental factors such as changes in river morphology, water flow patterns, and sedimentation may have impacted riverine habitats, reducing suitable areas for fishery activities. Additionally, human interventions such as dam construction, water diversion projects, and encroachment could have further exacerbated the decline in river fisheries' habitat availability.
2. **Beel fisheries:** The increase in water spread area from 100,815 hectares in 2009-10 to 71,843.50 hectares in 2022-23 indicates potential positive trends in beel fisheries. Beels, or seasonal floodplain wetlands, play vital roles as fish habitats and breeding grounds. The expansion of water spread areas in beels could be attributed to natural processes like

monsoonal flooding, as well as human interventions such as wetland conservation efforts and sustainable management practices.

3. **Forest fisheries:** Despite a slight increase in water spread area from 5,017 hectares in 2009-10 to 6,102.90 hectares in 2022-23, forest fisheries face challenges related to habitat degradation and fragmentation. Deforestation, land-use changes, and unsustainable fishing practices may have contributed to the marginal increase in forest fisheries' habitat area. Efforts to restore and conserve forested areas are crucial for maintaining and enhancing the ecological integrity of these habitats.
4. **Derelict water bodies/swamp:** The substantial increase in water spread area from 39,240 hectares in 2009-10 to 83,633.26 hectares in 2022-23 signifies positive developments in derelict water bodies and swamp ecosystems. Derelict water bodies, such as abandoned ponds and degraded wetlands, have the potential for restoration and reclamation. Initiatives focusing on wetland conservation, habitat restoration, and community-based management could have contributed to the expansion of water spread areas in these ecosystems.
5. **Reservoir fisheries:** The modest increase in water spread area from 1,713 hectares in 2009-10 to 3,096 hectares in 2022-23 suggests limited growth in reservoir fisheries. Reservoirs, created by dam construction for hydroelectricity and irrigation purposes, often face challenges related to habitat alteration, sedimentation, and water quality degradation. Sustainable reservoir management practices, including habitat enhancement, water quality monitoring, and fish stocking programs, are essential for optimizing the ecological and socio-economic benefits of reservoir fisheries.
6. **Ponds and tanks:** The significant increase in water spread area from 38,767 hectares in 2009-10 to 94,693.47 hectares in 2022-23 highlights the growing importance of ponds and tanks in Assam's fisheries sector. Ponds and tanks serve as critical fish production systems, offering opportunities for small-scale aquaculture and livelihood enhancement. Efforts to promote pond management practices, water conservation measures, and integrated aquaculture-agriculture systems have likely contributed to the expansion of water spread areas in ponds and tanks.

The changes in water spread areas of various types of fisheries in Assam underscore the complex interactions between natural processes, human activities, and management interventions. Addressing the challenges and harnessing the opportunities presented by these dynamic shifts are essential for ensuring the long-term sustainability and resilience of Assam's fisheries sector. Collaborative efforts involving government agencies, local communities, and

stakeholders are crucial for promoting ecosystem health, enhancing livelihoods, and achieving sustainable development goals in the fisheries sector.

Challenges faced by farmers:

Farmers in the fishery sector encounter a myriad of challenges throughout the production process, significantly impacting their livelihoods and the overall industry. These challenges can be broadly categorized into production-related, price-related, marketing-related, and miscellaneous issues.

Production-related problems encompass a range of difficulties, including inadequate technical assistance, scarcity of inputs, insufficient availability of subsidies, fluctuating demand for products, seasonal variations in demand, losses during handling, absence of proper packaging materials, and limited access to processing plants. These issues hamper the efficiency and productivity of fish farming operations, hindering farmers' ability to maximize their yields and profits

Price-related challenges, notably irregular payments, pose significant hurdles for fish farmers. Unpredictable payment schedules disrupt cash flows and financial planning, creating uncertainty and instability in the farming business. Such inconsistencies in payments can severely impact farmers' income and overall economic sustainability.

Marketing-related obstacles further exacerbate the challenges faced by fish farmers. Issues such as inadequate transportation facilities, disorganized markets, limited access to consumer markets, and distant marketplaces impede farmers' ability to effectively sell their produce. The prevalence of unorganized markets adds complexity to marketing efforts, making it difficult for farmers to establish stable market channels and secure fair prices for their products.

In addition to these primary challenges, fish farmers also grapple with supplementary issues. The lack of technical assistance for promotional activities, inadequate government support, and delays in subsidy disbursement emerge as prominent concerns. Without government assistance and subsidies, farmers struggle to access essential resources and support services, hindering their ability to operate efficiently and competitively in the market. Furthermore, the absence of technical support impedes the adoption of best practices and innovative techniques, limiting the potential for improved productivity and profitability. Overall, the cumulative effect of these challenges undermines the resilience and sustainability of the fishery sector, posing significant barriers to the growth and development of fish farming communities. Addressing these multifaceted issues requires collaborative efforts from policymakers, industry stakeholders, and support organizations to implement targeted interventions, improve infrastructure, streamline

market systems, and provide comprehensive support services to empower fish farmers and enhance their livelihoods.

Conclusion:

The fisheries sector in Assam is integral to the state's economic landscape, offering significant contributions to income generation, employment opportunities, and food security. Endowed with abundant aquatic resources, Assam's fisheries development is bolstered by its extensive river systems, floodplain lakes, and ponds, providing fertile grounds for growth and prosperity. Despite its importance, the fishery sector faces multifaceted challenges that impede its sustainable development. The changes in water spread areas of various types of fisheries underscore the dynamic nature of the sector, influenced by environmental, social, and economic factors. From declines in river fisheries to expansions in beel fisheries and derelict water bodies, these shifts highlight the need for adaptive management strategies to ensure the long-term viability of fishery resources.

However, alongside the opportunities lie formidable challenges faced by fish farmers throughout the production process. Production-related issues, such as inadequate technical assistance and scarcity of inputs, hamper productivity and efficiency. Price-related challenges, including irregular payments, disrupt financial stability and undermine economic sustainability. Marketing-related obstacles further exacerbate the difficulties, hindering farmers' ability to access markets and secure fair prices for their produce. Additionally, fish farmers grapple with supplementary challenges, such as the lack of government support and technical assistance, further impeding their ability to thrive in the sector. Addressing these challenges requires collaborative efforts from stakeholders across government, industry, and civil society to implement targeted interventions, improve infrastructure, and provide comprehensive support services to empower fish farmers and enhance their livelihoods.

To conclude we can say that while the fisheries sector in Assam holds immense potential for economic growth and development, overcoming the challenges requires concerted action and commitment. By addressing the multifaceted issues facing fish farmers and embracing sustainable management practices, Assam can unlock the full potential of its fisheries resources, ensuring prosperity for generations to come.

References:

1. Anonymous, (2023). Economic Survey Assam - (2023). Directorate of Economics and Statistics, Government of Assam. p. 396.
2. Anonymous, (2023). Statistical Handbook Assam - (2023). Directorate of Economics and Statistics, Government of Assam.

3. Bhattacharjya, B.K., Bhaumik, U., Sharma, A.P., (2017). Fish habitat and fisheries of Brahmaputra River in Assam, India. *Aquatic Ecosystem Health & Management* 20(1- 2), 102-115.
4. Bora, D. K., Gogoi, M., Gogoi, A. S., Bhuyan, R. P., Bharadwaj, E., Kalita, R., ... & Upadhyaya, S. (2024). Crush Tear Curl (CTC) Green Tea Processing: A Modified form of Green Tea for the Tea Lovers. *Journal of Scientific Research and Reports*, *30*(5), 535-546.
5. Borah, A., Gogoi, M., Dutta, P., Hazarika, B., Konwar, J., & Sahewalla, I. P. (2022). Assessing the Effects of Gas Flaring on the Growth Physiology of Tea Crop. *International Journal of Plant & Soil Science*, *34*(22), 1249-1255.
6. Borah, S. R., Dutta, P., Bhattacharjya, S., Konwar, M. J., Baruah, M., Bharali, A., ... & Chetia, S. K. (2020). Inclusion of seed production in rice-based cropping sequence as a means for doubling farmers' income. *Biological Forum – An International Journal*, *15(1): 237-242(2023)*.
7. Dutta, P., Chetia S. K., Hazarika, J., Sharma, R., and Deka, N. (2023). Economic impact of AAU rice varieties Ranjit and Bahadur in Assam. *Association of Rice Research Workers*
8. Dutta, P., Hazarika, B., Shyam, D., & Deka. N. (2023). Evolving Economic landscape: The changing role of Agriculture and Gender Dynamics in India. *Advances in Agricultural Extension.* ISBN: 978-93-55709-99-8
9. Dutta, P., Mahanta, N. J., Konwar, M. J., & Chetia, S. K. (2023). Assessing the Effectiveness of Climate-Resilient Rice Varieties in Building Adaptive Capacity for Small-Scale Farming Communities in Assam. *International Journal of Environment and Climate Change*, *13*(12), 607-613.
10. Hazarika, B., Dutta, P., & Gogoi. M. (2023). Exploring Assam's Tea Tourism: Opportunities and Obstacles. *Research Trends in Multidisciplinary Studies* (pp. 22-32). ISBN: 978-93-95847-60-5
11. Hazarika, B., Dutta, P., Gogoi, M., Gogoi, A. S., & Bora, D. K. (2024). Tea Tourism: Navigating the Future of Assam's Agritourism. *Journal of Scientific Research and Reports*, *30*(4), 77-88.
12. Hazarika, B., Gogoi, A.S. (2024). Unlocking The Carbon-Sequestering Potential Of Tea Gardens For Climate Change Mitigation. Advances in Agriculture Sciences. ISBN: 9789395847599. Page no. 94-104.
13. Hazarika, B., Gogoi, M. (2023). Harvesting Insights: Leveraging GIS and Remote Sensing for Modern Agriculture. Current Trends in Agriculture & Allied Sciences (Volume - 25) ISBN 9789359675992 Page no. 476-487

14. Hazarika, B., Gogoi, M., Boruah, K. P. (October (2023). Revolutionizing the Tea Industry with AI. Just Agriculture e-magazine ISSN. 2582-9149, Vol. 4 Issue- 2.
15. Hazarika, B., Kakoti, M., Talukdar, L., Saikia, B. (2024). Climate Change and Its Impact On The Global Tea Industry: A Comprehensive Exploration Of Cultivation, Economic Significance, And Adaptation Strategies. Research and Reviews in Literature, Social Sciences, Education, Commerce and Management ISBN: 9789395847803
16. Nandi *et al.*, 2022. Fisheries in Assam: Status and Way Forward. Biotica Research Today 4(11):768-770.

INSECT BEHAVIOUR

Swarupa Balasaheb Jadhav

Department of Zoology,

Rajarshi Shahu College (Autonomous), Latur, M.S.

Corresponding author E-mail: swarupaspatil.123@gmail.com

Abstract:

Environmental hazards such as temperature, humidity, parasites and toxins covers a very wide range of activities, including locomotion, grooming, feeding, communication, reproduction, dispersal, flight, learning, migration, host or prey selection understanding the behavior of pest and beneficial insects could improve pest-management programs.

Keywords: Insect Behavior, Social Insect, Social Insect.

Introduction:

An insect behavior refers to the various actions of an insect in response to stimulus or to its environment. Inside moths which show positive phototoxic effect i.e. they tend to fly towards the source of light. Insect behaviors inside moths affect by environmental cues like temperature, humidity and toxins.

Ants walking in a line and following one another they are really following a trail of pheromones that each one in leaving behind to show where to go this is due to as ants release chemical that affect the behavior of the other members of the same species.

Wide range of activities are covered by insect behavior such as feeding, locomotion, grooming, reproduction, learning, migration and communication.

Appearance and habits:

Majority of insects varied in size some are as small as 6 mm (0.2 inch) while some are as large as 27 cm for example feather winged beetles and parasitic wasps are microscopic while Hercules beetles, African goliath beetles certain Australian stick insects and wingspan of Hercules moth can be large as 27 cm.

Types of insect behavior:

There are two types of insect behavior observed in animals

1) Innate behavior and

2) Learned behavior

Moth is parallel to the ground because this is due to tendency of the moth to keep the sun overhead but this is unclear behavior of moth.

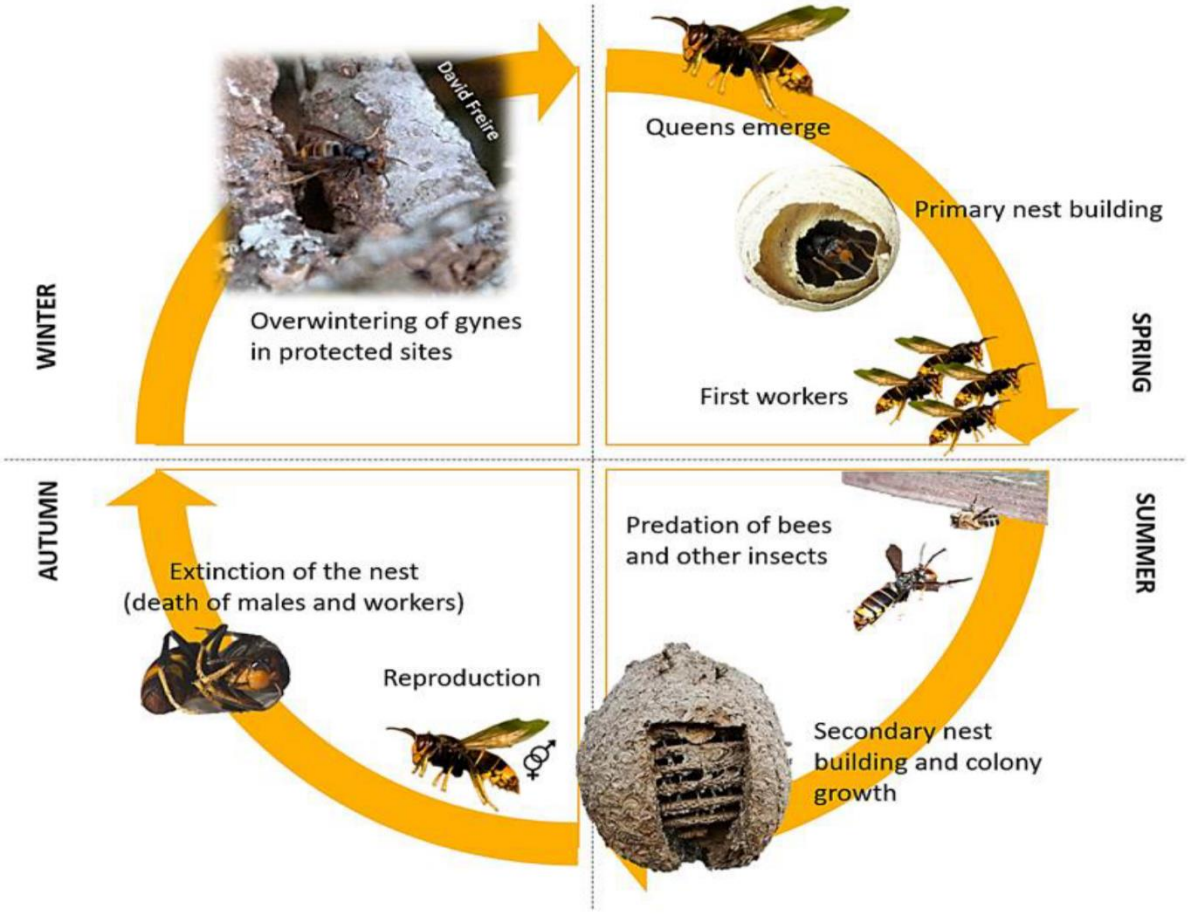


Figure 1: Types of Insect Bahavior

Inside insect Honey bee learn behavior is observed they are able to learn from environmental signals used to locate food source this learn behavior acquires in animal through experience or learning.

Types of insect behavior:

1) Social insect:

Any category of insects where one of the kinds interacts with the rest of the members of some species are considered social.

Many social behavior like feeding, aggregation communal nest sites, parental care of adults for younger one this type of social behavior observed in many types of insects like bees, ants and many such other animals.

According to rule by emotologists, few of these behaviors are not considered as true social insect.

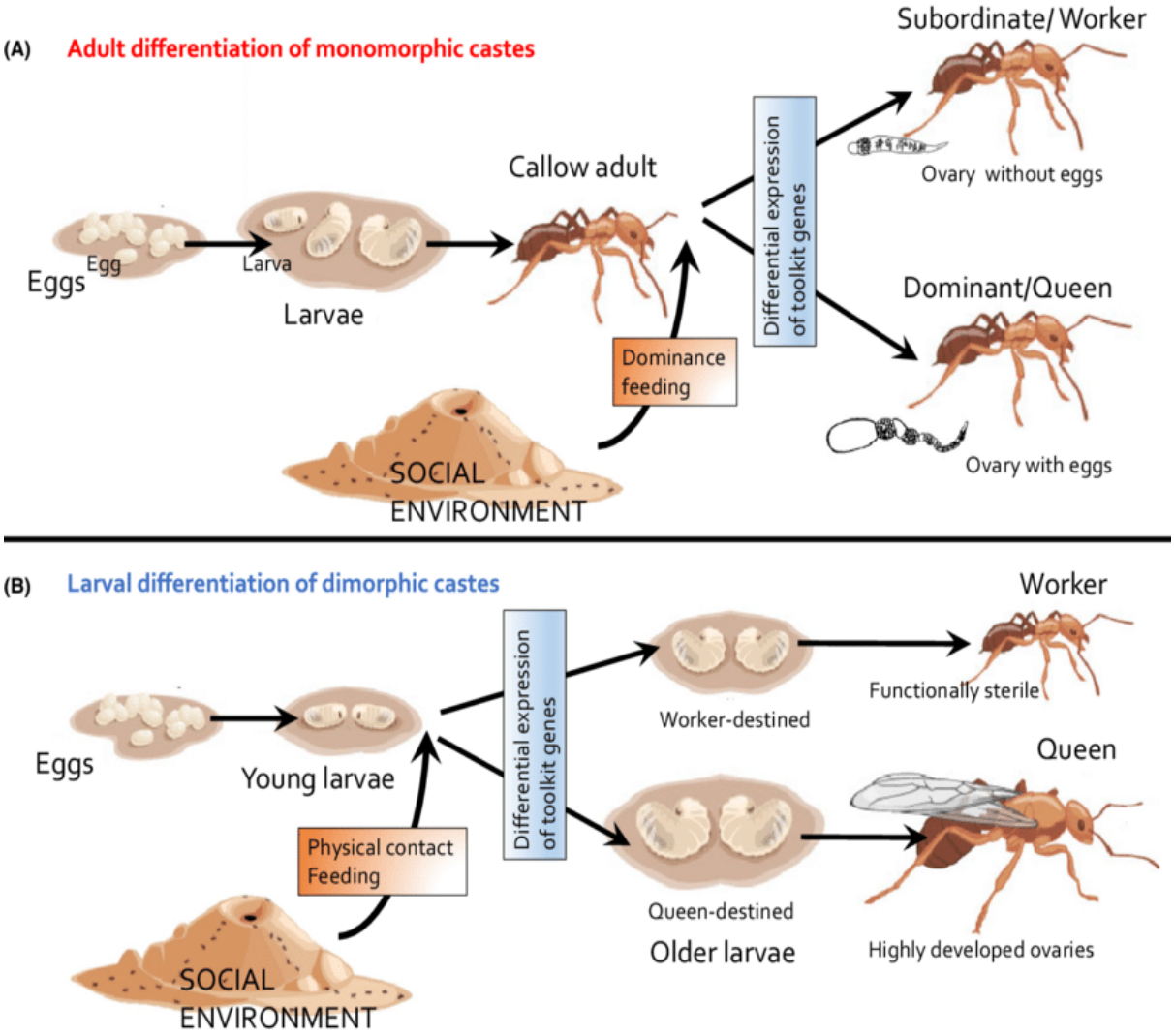


Figure 2: Social insect diagram

Unsociality exhibits characters like sharing common nest site, individuals of same species contributes equally in caring for an offspring overlapping of the generations within the community means young's are involved as working force of the community.

Unsociality is the highest level of social culture.

Formica Yessensis forms megacolony with 45,000 interconneccting nests with millions of queens and more than 405 million of steriles they cover the area about 2.7 kilometers. Formica Yessensis habitat is in Japan.

Types of social insect:

1) Quasi- social insect:

Example of Quasi-social insect are arched bees. Where females share the nest and care and protect their little ones together. Quasi-social insects are little more advanced socially than

of sub-social insect's quasi-social insects exhibit cooperative parental care for offspring of the family.

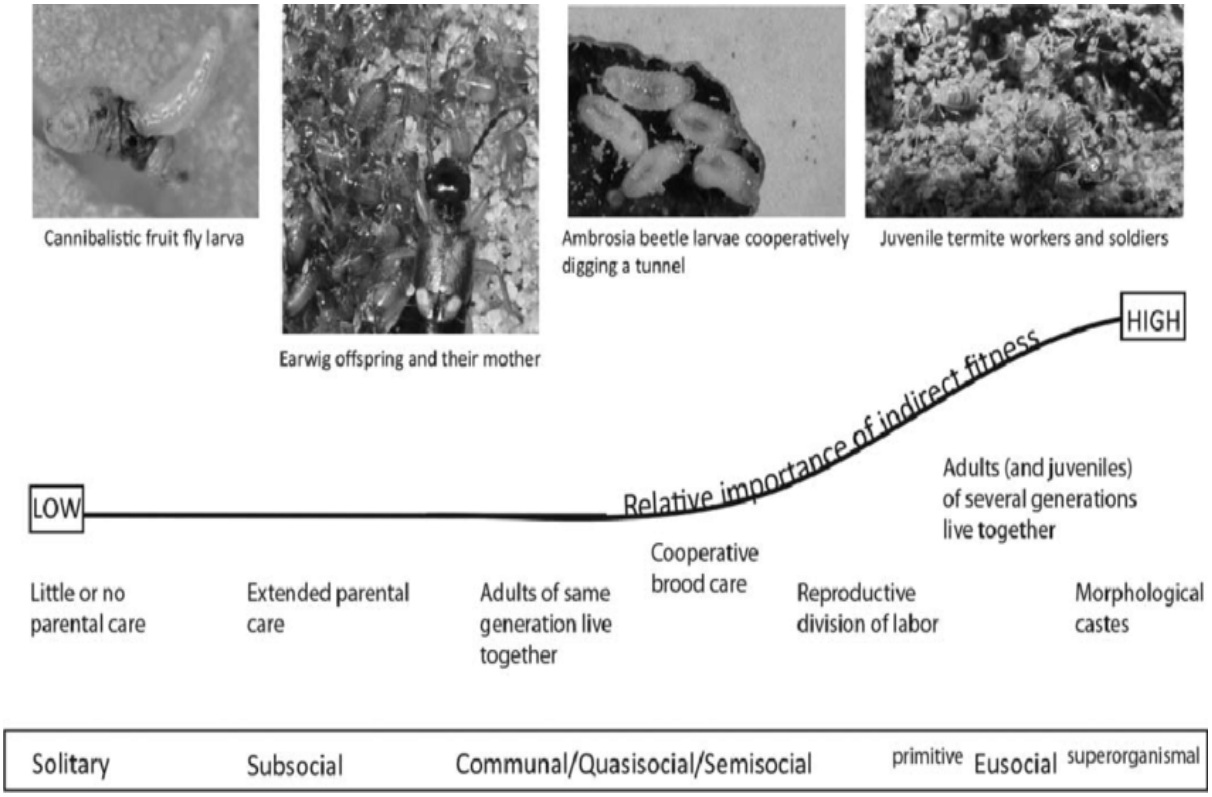


Figure 3: Quasi-Social insect

2) Semi-social insect:

Common example of semi-social insect is paper warps in paper moth nonproductive workers as well as productive workers help in expanding and building new nests for young-common feature for this adult stage of insect leave the nest when the offspring arrives to build a new nest.

Semi-social insects are similar to quasi-social insect in terms of they have nonreductive workers, support and protect to their broods in single nest.

Difficulty in defining the behavior in there is difficulty in genetic analyses of behavior this is due to multiple complication.

Due to confusion regarding number of genes involved there is difficulty in defining the behavior in clear manner difficulty in distinguishing between behavior and physiology behavior can be examined in four different viewpoints.

1) Cause of behavior (or control)
2) Behavior development during individuals lifespan
3) Behavior function
4) How the behavior evolved. (Wyatt, 1997)

Genetic analyses of insect behavior require carful control of environmental conditions, because even subtle differences in test conditions can influence results of assays. (Vanin et at. 2012).

Figure 4: Semi-Social insect

Behavior genetics was limited to demonstrating that behavioral trait was heritable, determining whether its mode of inheritance was dominant or recessive, sex-linked or autosomal and resolving whether variation was due to single or multiple genes. Behavior genetics began to develop as a field of study in 1960s.

The genetic analysis of behavior right-fully has been perceived to be more complex than analysis of morphological or anatomical traits (Vanin et.as.2012).

Using honey bees, grasshoppers, honey bees, Nasonia parasitoids and crickets, drosophila melanogaster, the genetic basis of insect behavior analyzed.

Primitively eusocial insects:

The scavengers or sterile group are all the same with very minute or no morphological differences as in primitive eusocial insects all are productive.

For example:

Bumblebees being considered as primitively eusocial as the size of queen bee is slightly bigger than size of sterile casts.

References:

1. Caetano, D. S., & Aisenberg, A. (2014). Forgotten treasures: The fate of data in animal behavior studies. *Animal Behaviour, 98*, 1-5.
2. Dunn, R. R. (2005). Modern insect extinctions, the neglected majority. *Conservation Biology, 19*, 1030-1036.
3. Forrest, J. R. (2016). Complex responses of insect phenology to climate change. *Current Opinion in Insect Science, 17*, 49-54.
4. Greene, H. W. (2005). Organisms in nature as a central focus for biology. *Trends in Ecology and Evolution, 20*, 23-27.
5. Haywood, B. K. (2014). A "Sense of place" in public participation in scientific research. *Science Education, 98*, 64-83.
6. Klein, B. A., & Seeley, T. D. (2015). The declining use of animal and behavior images in animal behaviour journals. *Animal Behaviour, 103*, 171-177.
7. Rehan, S. M., & Toth, A. L. (2015). Climbing the social ladder: The molecular evolution of sociality. *Trends in Ecology and Evolution, 30*, 426-433.
8. Webster, M. S. (2017). *The extended Specimen: Emerging Frontiers in Collections-based Ornithological Research*. CRC Press.

AN EXTENSIVE VIEW ON DIETARY NEEDS OF MULTIPLE MYELOMA FRUIT BROWNIES

Poovizhi Selvi Ravi* and Anitha Ramagupandian

Department of Nutrition and Dietetics,

K.S.R College of Arts and Science for Women,

Tiruchengode – 637215, Namakkal, Tamilnadu, India

*Corresponding author E-mail: poomphil@gmail.com

Abstract:

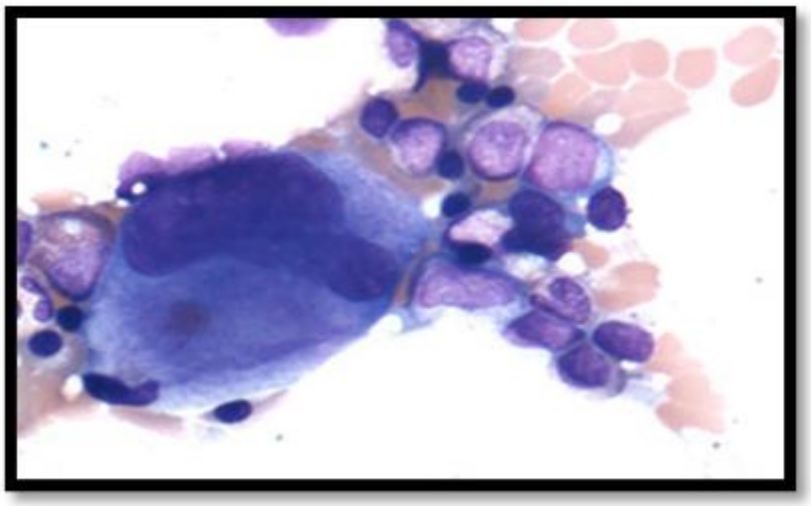
Multiple Myeloma (MM) is a cancer that forms in a type of white blood cell called a plasma cell. Healthy plasma cells help fights infections by making proteins called antibodies. Antibodies find and attack germs. The cause of monoclonal gammopathies and the mechanisms of progression are unknown. The diagnosis of Multiple Myloma (MM) requires the presence of an M-protein in serum and/or urine, increased bone marrow plasma cells and related organ or tissue impairment. Cytogenetic status, serum b2-microglobulin and response to therapy are the key prognostic factors. He most common presenting symptoms of Multiple Myloma are fatigue and bone pain. Anaemia occurs in approximately 75% of patients and contributes to fatigue. Osteolytic skeletal lesions can be detected in approximately 80% of patients. Other common findings at presentation include hypocalcaemia (15%) and elevated serum keratinises level (≥2 mg/dL) (20%). Rice flakes demand is growing globally, edible flakes market size has been valued about USD 17.45 million in 2020 and is predicted to grow more and more in coming days. The Rice flakes were made with incorporated in pomegranate which are rich in minerals and antioxidants helps in avoiding cellular damage in human body. The proximate analysis revealed that the carbohydrate is 84.29%, protein is 15.75%, total fat is 4.96%,acidity of extracted fat is 0.9164% total ash is 0.72% and acid insoluble ash was 0.063% per 100g of the sample.

Keywords: Allergenic stem cell transplantation, B2-Microglobulin, Monoclonal Gammopathy, Multiple Myeloma, rice flakes and pomegranate brownie, etc.,

Introduction:

Myeloma is a blood cancer of cells found in the bone marrow, specifically the so-called “plasma cells.” The bone marrow is the spongy tissue inside your bones that normally creates the different parts of your blood. Plasma cells are a key part of the body’s immune system. They

produce antibodies that help the body fight infection. Myeloma begins when healthy plasma cells change and grow out of control. This may result in multiple bone lesions that increase the risk of bone fractures.



Definition

Multiple myeloma is cancer that starts in plasma cells in your bone marrow. This is the soft, inner part of some bones where new blood cells are made. Plasma cells are part of your immune system. They make proteins called antibodies to help fight infections and diseases. Plasma cells are found mainly in the bone marrow.

Also, mutated plasma cells go through a series of changes. This can eventually cause excess cell growth. It can also cause tumors to form in your bones. Along with bone tumors, also cause other health problems. These include having too much calcium in your blood, low blood cell counts, kidney problems, and frequent infections.

Signs and symptoms

- Constipation.
- Loss of appetite.
- Mental fogginess or confusion.

Causes

Multiple myeloma begins with one plasma cell in the bone marrow. The bone marrow is the soft matter inside bones where blood cells are made. Something happens that turns the plasma cell into a cancerous myeloma cell. The myeloma cell begins making a lot more myeloma cells quickly.

Healthy cells grow at a set pace and die at a set time. Cancer cells don't follow these rules. They make a lot of extra cells. The cells continue living when healthy cells would die. In

myeloma, the cancer cells build up in the bone marrow and crowd out the healthy blood cells. This leads to tiredness and not being able to fight infections.

Risk factors

Factors that may increase the risk of multiple myeloma include:

- **Getting older.** Most people are diagnosed in their late 60s.
- **Being male.** Men are more likely to develop the disease than are women.
- **Being Black.** Black people are more likely to develop multiple myeloma than are people of other races.

Treatment

Multiple myeloma treatment isn't always needed right away. If there are no symptoms, you might have tests to watch the myeloma to see if it gets worse. When multiple myeloma causes symptoms, treatment often starts with medicine. Treatment can help relieve pain, control complications, and slow the growth of the myeloma cells

Medical therapy for multiple myeloma

Treatments might include:

Targeted therapy. Targeted therapy uses medicines that attack specific chemicals in the cancer cells. By blocking these chemicals, targeted treatments can cause cancer cells to die.

Immunotherapy. Immunotherapy is a treatment with medicine that helps the body's immune system to kill cancer cells. The immune system fights off diseases by attacking germs and other cells that shouldn't be in the body. Cancer cells survive by hiding from the immune system. Immunotherapy helps the immune system cells find and kill the cancer cells..

Nutrition therapy for multiple myloma

Although no specific diet can treat multiple myeloma, certain nutrition strategies can help a person to increase the white blood cells level. There are no proven special diets to treat multiple myeloma. However, there are specific nutrition strategies that can be used to treat common symptoms, such as kidney damage and anemia, as well as to reduce cancer recurrence.

Despite the lack of evidence to support an alternative diet, proper nutrition with a focus on particular foods still plays a role in the overall health, energy levels, and strength of people with multiple myeloma.

Benefits of the rice flakes with pomogranet brownie for multiple myeloma:

Combines rice flakes and pomegranate for a delightful dessert or snack. Rich in protein, calcium, iron, fiber, and vitamin C. Cocoa powder, dark chocolate, and peanuts may help reduce stress. Keeps you feeling full and may reduce daily calorie intake. Good source of protein from

peanuts and iron from pomegranate, dates, chocolate, and jaggery. A delicious way to treat yourself with health benefits.

Improvement from the complications

Rice flakes with pomegranate brownies, for children with sickle cell disease causes faster red blood cell breakdown, burning more energy. Special high-calorie brownies address this energy gap for children with sickle cell disease. Brownies are packed with iron, protein, fiber, and minerals. They see a 50% improvement in consumption due to appealing ingredients.

Development of rice flakes and pomogranate fruit brownie (RPF)

Introduction

A Rice flakes and Pomogranate (RPF) Fruit Browine or simply a brownie is a chocolate baked confection. Brownies come in a variety of forms and may be either fudgy or cakey, depending on their density. Brownies often, but not always, have a glossy "skin" on their upper crust referred to as flint. They may also include nuts, frosting, chocolate chips, or other ingredients. A variation made with brown sugar and vanilla rather than chocolate in the batter is called a blond brownie or blondie. The brownie was developed in the United States at the end of the 19th century and popularized there during the first half of the 20th century.

Methodology:

This recipe incorporates rice flakes, pomegranate, gooseberry, and dark chocolate, each offering unique advantages. Rice flakes provide a light, crunchy texture and easily digestible carbohydrates for sustained energy, without spiking blood sugar due to their low sugar and fat content. Pomegranate adds a burst of flavor and antioxidants, potentially reducing inflammation. Dark chocolate contributes essential minerals like magnesium and iron, while gooseberry, rich in vitamin C, boasts potential antioxidant and anti-inflammatory properties. Together, these ingredients create delicious and nutritious brownies.

Procurement of raw materials

The raw materials such as All-purpose flour, Pomegranate, Dates, Groundnut, Brown sugar, Dark chocolate, Butter, Coco powder, Baking soda, Yeast, Rice flakes. These ingredients are purchased in the Kannan Departmental store which was situated in kadachanellur, Erode District, Tamilnadu.

Table 1: Standardization of the RPF brownie:

Ingredients	X1	X2	X3
Rice flakes (gm)	9	7	7
Pomegranate (gm)	8	9	9
Dates (gm)	2	2	2
Banana (gm)	2	2	2
Jaggery – sugar cane (gm)	9	8	7
Dark chocolate (gm)	5	7	7
Groundnut (gm)	5.5	5.5	5.5
All purpose flour (gm)	8	8	9
Butter (gm)	1	1	1
Gooseberry (gm)	0.5	0.5	0.5

Flow chart of development of RPF brownie

(1) Procurement of dry ingredients

↓

(2) Mixing of dry ingredients as per formulation

↓

(3) Transfer batter to greased tray

↓

(4) Mixing of batter to a uniform consistency and leave it for 15 minutes and placed the tray in the oven and bake it for 20 minutes

↓

(5) Cut the baked brownie into pieces and serve

Nutrition calculation

(Calculation Method-NIN, Revised -1989, Reprint -2018):

Table 2: Nutritional evaluation of RPF

Ingredient	Quantity (g)	Energy (kcal)	Protein (gm)	Carbohydrate (gm)	Fat (gm)	Fibre (gm)	vitamin - C (mg)	Iron (mg)
Rice Flakes	15	51.9	0.99	11.5	0.18	0.105	0	3
Pomegranate	20	13	0.79	2.9	0.02	1.02	3.2	0.358
Black Dates	5	15.8	0.125	3.79	0.02	0.19	0.15	0.73
Country Banana	10	11.6	0.12	2.7	0.03	0.04	0.7	0.036
Jaggery (Sugar cane)	5	17	0.54	4.75	0.005	-----	0.35	0.132
Dark Chocolate	10	50	0.49	5.96	3.42	0.6	0	0.8
Roasted Groundnut	5	28.5	1.91	1.33	1.9	0.15	0	0.155
All purpose Flour	10	36	1.88	7.63	0.1	0.3	0	0.24
Butter	10	72.9	-----	-----	8.1	----	----	----
Gooseberry	10	5.3	0.188	1.11	0.02	0.32	4.9	0.2
Total		**304**	**6.84**	**41.69**	**13.795**	**2.725**	**9.3**	**5.651**

The nutritive value Calculated by NIN book revised copy for the year of 1989 and reprinted was 2018. We have calculated the value for the Rice flakes and Pomogranate (RPF) Fruit Browine, provides Energy – 304 kcal, Protein – 6.84g, Carbohydrates – 41.69g, of fat, 2.725g Fibre – 13.795g, Vitamin C – 9.3mg and Iron – 5.651mg accordingly.

Organoleptic evaluation:

The brownies produced from all purpose flour was tested for evaluation of sensory characteristics and acceptability. This measured the acceptability of the characteristics of the product in comparison to percentage of all purpose flour that it contains and identify which percentage variation is the most acceptable. Six students were randomly selected through convenience semi trained panel sampling to participate in the study also four faculties were selected as trained panels, and they were asked to accomplish informed consent forms. The sensory evaluation forms used a 5-point hedonic scale for the general attributes such as and texture taste, appearance, aroma. The best and least sample were also asked in the latter part of the questionnaire along with recommendations and suggestions. A trained panel consists of experts who have undergone extensive training and calibration to evaluate specific sensory attributes of food using standardized methods and scale.

Trained panels:

Here, the above panels are from the K.S.R College of Arts and Science for Women, Trichengode, Namakkal, Tamilnadu, faculties in the Department of Nutrition and Dietetics, selected as a trained panels.

Table 3: Organoleptic evaluation

Panels Name	Texture	Appearance	Aroma	Flavour	Overall Acceptability
Trained Panels					
Indra N	5	5	5	4	5
Logesheari S	5	4	5	5	5
Swathy S	5	4	5	5	5
Poovizhi Selvi R	4	5	5	4	5
Semi Trained Panels					
Dhanusha N	5	4	5	5	5
Raveena A	5	5	4	4	5
Vedhavithya P	5	4	5	5	5
Yuva Sri M	5	5	5	4	4
Pavithra M	5	4	5	5	5
Madhumitha R	5	5	5	5	5

Semi trained panels:

This type of panels should be constituted from persons normally familiar with quality of milk and different classes of dairy products. This panel is capable of discriminating differences and communicating their reactions, though it may not have been formally trained.

The above mentioned table, the semi trained panels are from the K.S.R College of arts and science for women, Trichengode, Namakkal, Tamilnadu, Students in the department of Nutrition and Dietetics, selected as a Semi trained panels.

Nutrient analysis of the standardised RPF brownie

The nutrients namely moisture, ash, protein, iron, Of the product were analysed different adopted method that was mentioned in the table.

Table 4: Nutrient content of the standardised RPF brownie (per 100g)

Nutrients	Rpf Brownie (100g)	Method
Moisture (%)	22%	Gravimetric method (1935)
Ash (g)	0.7(g)	Dry ash test (1976)
Protein (g)	6.5 g	Ninhydrin test (1910) Biuret test (1833)
Iron (mg)	6.3 mg	Calorimetric test (1934)

In this table, were we analysed the moisture content - 22% in the method of gravimetric method (1935). Ash content - 0.7g of dry ash method (1976). Protein - 6.5g in the method of ninhydrin test (1910) & Biuret test (1833) and the iron - 6.3mg in the calorimetric test (1934).

Results and Discussion:

In the brownie production, three trials were done to account different comments and suggestions from evaluators. The use of the all-purpose flour and dark chocolate flour ratio was also identified from predetermined percentages. The needed amount for the recipe is one-fourth cup, which is equivalent to 4 tablespoons, so the amount of substitution is identified using this unit. The variation needed rice flakes, pomegranate, dates, banana, jeggery, dark chocolate, groundnut, all pupose flour, and butter were selected In different ration for the development of the product.

The results of sensory evaluations can be used to make informed decisions about food product development, quality control, and marketing. For example, if a manufacturer finds that consumers prefer a new product with a sweeter taste, they can adjust the formulation accordingly. Sensory evaluation notes the human attires like taste, texture, flavour, appearance of

the humans. The formulation of Rice flakes with pomogranet Brownie provides nutritious value to the product.

Conclusion:

Today's health-conscious consumers are driving the demand for healthier bakery options. Producers are responding with innovative snacks packed with beneficial nutrients like iron and protein. Rice Flakes with Pomegranate Brownies are a prime example, offering a delicious and nutritious alternative to traditional brownies. Rich in iron and protein, these brownies are a perfect choice for those seeking a healthier treat, which is the best supplemented for the selected disease. In these the presence of nutrients may help the tissues damage and replacing the muscle mass. And also its very suitable for all age group people who has suffered in this disease, particularly it helps to maintain good health in nature.

References:

1. Blommestein, H. M., van Beurden-Tan, C. H. Y., Franken, M. G., Uyl-de Groot, C. A., Sonneveld, P., & Zweegman, S. (2019). Efficacy of first-line treatments for multiple myeloma patients not eligible for stem cell transplantation: A network meta-analysis. *Haematologica, 104*(5), 1026-1035.
2. Chesi, M., Nardini, E., Lim, R. S., Smith, K. D., Kuehl, W. M., & Bergsagel, P. L. (1998). The t(4;14) translocation in myeloma dysregulates both FGFR3 and a novel gene, MMSET, resulting in IgH/MMSET hybrid transcripts. *Blood, 92*(9), 3025-3034.
3. Dhodapkar MV. MGUS to myeloma: a mysterious gammopathy of underexplored significance. *Blood.* 2016 Dec 8;128(23):2599-2606. doi: 10.1182/blood-2016-09-692954.
4. Kiss, S., Gede, N., Soós, A., Hegyi, P., Nagy, B., Imrei, M., Czibere, B., Farkas, N., Hanák, L., Szakács, Z., Erőss, B., & Alizadeh, H. (2021). Efficacy of first-line treatment options in transplant-ineligible multiple myeloma: A network meta-analysis. *Critical Reviews in Oncology/Hematology, 168*, 103504.
5. Mateos, M. V., & Landgren, O. (2016). MGUS and Smoldering Multiple Myeloma: Diagnosis and Epidemiology. *Cancer Treatment and Research, 169*, 3-12.
6. Pasca, S., Tomuleasa, C., Teodorescu, P., Ghiaur, G., Dima, D., Moisoiu, V., Berce, C., Stefan, C., Ciechanover, A., & Einsele, H. (2019). KRAS/NRAS/BRAF Mutations as Potential Targets in Multiple Myeloma. *Frontiers in Oncology, 9*, 1137.

AN EXTENSIVE REVIEW ON DIETARY NEEDS OF SICKLE CELL ANEMIA AN IRON SUPPLEMENTED BROWNIES

Poovizhi Selvi Ravi* and Jeeva Muthu
Department of Nutrition and Dietetics,
K.S.R College of Arts and Science for Women,
Tiruchengode – 637215, Namakkal, Tamilnadu, India
*Corresponding author E-mail: poomphil@gmail.com

Abstract:

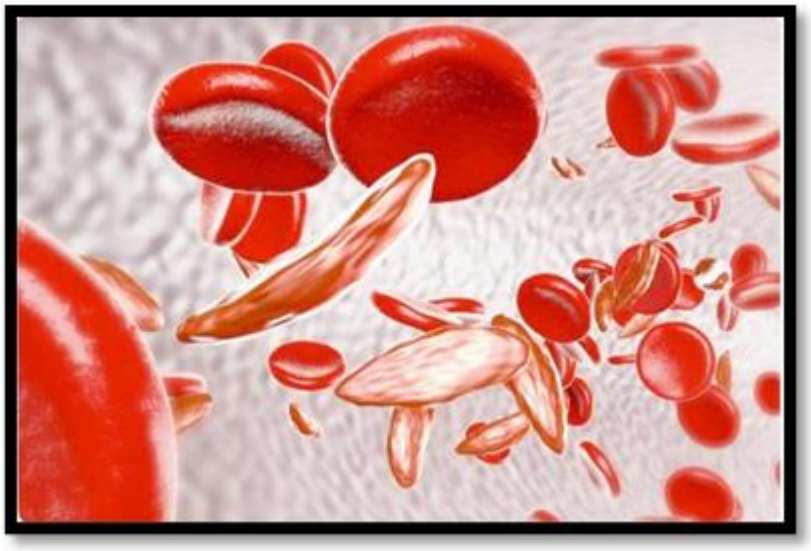
Sickle cell disease is a serious problem in Africa where the required medication is often not available or too expensive for local Africans. Other possibilities have to be investigated, like traditional medication in the form of nutritional rich foods to control the symptoms of the disease. However, the growing knowledge that nutrition underlies disease severity has sparked interest in promoting dietary supplementation to treat these patients. This review aims to highlight the fact that children and adults with sickle cell disease require much higher amounts of energy and protein (larger macronutrient intake) than healthy and predisposed individuals. Become malnourished if energy intake is consistently low. Deficiencies may also exist for micronutrients, such as glutathione, which has both anti-inflammatory and antioxidant properties. Chronic inflammation and oxidative stress are central issues that increase the severity of sickle cell disease. In conclusion, it is essential to devote more effort and resources to establishing the Recommended Dietary Reference Intake (DRI)/Recommended Dietary Allowance (RDA) for patients with SCA and can Nutritional intervention should be included as an adjunctive treatment alongside standard practice.

Keywords: Sickle Cell Disease, Anti-Inflammatory, Iron Supplemented Brownie,

Introduction:

Sickle cell disease is a lifelong blood disorder characterized by red blood cells that have a rigid and abnormal sickle shape. Sickle cell disease reduces cell mobility and carries the risk of various complications. Sickle disease is caused by mutations in the hemoglobin gene. The average life expectancy for men and women is 42 and 48 years respectively. Sickle cell disease, which usually manifests in childhood, occurs more frequently in people from tropical and subtropical regions affected by malaria, because entry of malaria plasmodium is prevented. By

the spread of the cells it infects. According to the National Institutes of Health, the incidence in the United States is about 1 in 5,000 primarily affecting African Americans.



Definition:

Sickle cell disease is an inherited blood disorder with abnormally shaped red blood cells that cause blockages and are less durable. It's most common in people of West and Central African

Signs and symptoms:

Sickle cell disease is an inherited blood disorder that affects the shape of red blood cells, causing them to become sickle/crescent shaped. Symptoms of sickle cell disease vary between individuals, but some of the commonly noted symptoms include:

- Anemia or decreased number of RBCs and oxygen
- Irritability or fussiness in babies
- Severe fatigue or tiredness

Causes:

- Caused by abnormal hemoglobin (hemoglobin S) in red blood cells
- Hemoglobin S makes red blood cells sickle-shaped (rigid and sticky)
- Sickle cells clog capillaries (smallest blood vessels)

Risk factors:

- Inherited blood disorder affecting red blood cells
- Sickle-shaped red blood cells are stiff, sticky and die prematurely (10-20 days) compared to normal cells (120 days)
- Early death of red blood cells leads to chronic anemia (low oxygen transport)

Treatments:

Treatments for sickle cell anemia aim to manage symptoms and prevent complications. Medications such as Ibuprofen, Hydroxyurea, and L-glutamine are used to reduce pain during crises, reduce episodes of pain, and reduce acute complications of the disease, respectively. Adakveo (crizanluzumab), Casgevy (exagamglogene autotemcel), Endari (L-glutamine), Hydroxyurea

Diet and nutrition:

Children with sickle cell disease need a balanced diet just like any growing child. This means plenty of fruits, vegetables, whole grains, and protein sources like fish, meat, or beans. If your family is vegetarian, a nurse can help ensure your child gets enough protein and healthy fats. While children with sickle cell disease don't require a special diet, they are more prone to infections. Be sure to cook foods like chicken and eggs thoroughly to avoid salmonella poisoning.

Development of iron supplemented brownie

A delightful American invention, brownies are baked chocolate treats loved for their rich flavor and chewy texture. They come in fudgy or cakey varieties, and often have a glossy top crust. Bakers love to add nuts, frosting, or chocolate chips for extra indulgence. For those who prefer a lighter taste, there are blondes – made with brown sugar and vanilla instead of chocolate. Brownies became popular in the late 19th century and are enjoyed everywhere from cafes to ice cream parlors. They are a favorite homemade treat, and every baker has their own special touch to create the perfect brownie.

Methodology:

This recipe incorporates rice flakes, pomegranate, gooseberry, and dark chocolate, each offering unique advantages. Rice flakes provide a light, crunchy texture and easily digestible carbohydrates for sustained energy, without spiking blood sugar due to their low sugar and fat content. Pomegranate adds a burst of flavor and antioxidants, potentially reducing inflammation.

Dark chocolate contributes essential minerals like magnesium and iron, while gooseberry, rich in vitamin C, boasts potential antioxidant and anti-inflammatory properties. Together, these ingredients create delicious and nutritious brownies.

Processing of raw materials:

The recipe begins with preparing dry ingredients (shelled peanuts, flour, rice flakes, and dates) and melted dark chocolate. Pomegranate seeds are separated and juice is reserved. Wet ingredients are combined with pomegranate juice, while dry ingredients are mixed with rice flakes, flour, gooseberry, dates, and peanuts. The mixture is blended into a batter and baked in a preheated oven for 10-20 minutes. The finished brownie is dusted with cocoa powder.

Standardization of the product:

Ingredients	X1	X2	X3
Rice flakes (gm)	9	7	7
Pomegranate (gm)	8	9	9
Dates (gm)	2	2	2
Banana (gm)	2	2	2
Jaggery (cane) (gm)	9	8	7
Dark chocolate (gm)	5	7	7
Groundnut (gm)	5.5	5.5	5.5
All purpose flour (gm)	8	8	9
Butter (gm)	1	1	1
Gooseberry (gm)	0.5	0.5	0.5

Flow chart of development of brownie:

Procurement of dry ingredients

↓

↓

Mixing of dry ingredients as per formulation

↓

Transfer batter to greased tray

↓

Mixing of batter to a uniform consistency and leave it for 15 minutes and placed the tray in the oven and bake it for 20 minutes

↓

Cut the baked brownie into pieces and serve

Results and Discussion:

The results of sensory evaluations can be used to make informed decisions about food product development, quality control, and marketing. For example, if a manufacturer finds that

consumers prefer a new product with a sweeter taste, they can adjust the formulation accordingly. Sensory evaluation notes the human attires like taste, texture, flavor, appearance of the humans. The formulation of Rice flakes with pomegranate Brownie provides nutritious value to the product.

Proximate nutrient analysis (Calculation method - NIN, Revised -1989, Reprint -2018):

Ingredient	**Quantity (g)**	**Energy (kcal)**	**Protein (g)**	**Carbohydrate (g)**	**Fiber (g)**	**Vitamin - C (mg)**	**Iron (mg)**
Rice flakes	15 g	51.9l	0.99	11.5	0.105	-	3
Pomegranate	20 g	13	0.79	2.9	1.02	3.2	0.358
Dates	5 g	15.8	0.125	3.79	0.19	0.15	0.73
Banana	10 g	11.6	0.12	2.7	0.04	0.7	0.036
Jaggery (cane)	5 g	17	0.54	4.75	-	0.35	0.132
Dark chocolate	10 g	50	0.49	5.96	0.6	-	0.8
Groundnut	5 g	28.5	1.91	1.33	0.15	-	0.155
All purpose flour	10 g	36	1.88	7.63	0.3	-	0.24
Butter	10 g	72.9	-	-	-	-	-
Gooseberry	10 g	5.3	0.188	1.11	0.32 g	4.9 mg	0.2
Total		**304 kcal**	**6.84 g**	**41.69 g**	**2.725 g**	**9.3 mg**	**5.651 mg**

The analysis for Rice flakes with pomegranate brownie is Energy - 304 kcal, Protein - 6.84 g , Carbohydrate - 41.69 g, Fibre - 2.725 g, Vitamin C - 9.3 mg, Iron - 5.651 mg respectively.

Sensory evaluation:

Food sensory testing involves the use of human sense in the objective evaluation is done with 5 point hedonic scale to understand the acceptability of the product. Sensory evaluation is done to the developed Rice flakes with pomegranate brownie 3 trained panelist and 6 semi trained panelist from KSR College of arts and science for women, Tiruchengode has done the sensory evaluation. The product is evaluated for its appearance, colour, taste, texture and overall acceptability and this standardized Rice flakes with pomegranate brownie is selected for further analysis.

The scores are given in the below table:

Proximate analysis:

Benefits of the rice flakes with pomegranate brownie for sickle cell disease:

Combines rice flakes and pomegranate for a delightful dessert or snack. Rich in protein, calcium, iron, fiber, and vitamin C. Cocoa powder, dark chocolate, and peanuts may help reduce stress. Keeps you feeling full and may reduce daily calorie intake. Good source of protein from peanuts and iron from pomegranate, dates, chocolate, and jaggery. A delicious way to treat yourself with health benefits.

Improvement from the complications:

Rice Flakes with Pomegranate Brownies, For Children with Sickle Cell Disease causes faster red blood cell breakdown, burning more energy. Special high-calorie brownies address this energy gap for children with sickle cell disease. Brownies are packed with iron, protein, fiber, and minerals. They see a 50% improvement in consumption due to appealing ingredients.

Panels Name	Appearance	Texture	Taste	Flavour	Overall Acceptability
Trained Panels					
N Indra	5	5	5	4	5
S Logeshwari	5	4	5	5	5
S Swathy	5	4	5	5	5
Poovizhi Selvi R	4	5	5	4	5
Semi Trained Panels					
N Dhanusri	5	4	5	5	5
A Raveena	5	5	4	4	5
P Vedhavithya	5	4	5	5	5
M Yuva sri	5	5	5	4	4
M Pavithra	5	4	5	5	5
R Madhumitha	5	5	5	5	5

Nutrient composition of the standardized rice flakes with pomegranate brownie:

The nutrients namely moisture, ash, protein, iron of the standardized rice flakes with pomegranate brownie were analysed and the results are presented in table

Nutrient content of the standardized rice flakes with pomegranate brownie (per 100g):

Nutrients	Brownie X3 (100g)	Method
Moisture (%)	22 %	Gravimetric method (1935)
Ash (g)	0.7 g	Dry ash test (1976)
Protein (g)	6.5 g	Ninhydrin test (1910) Biuret test (1833)
Iron (mg)	6.3 mg	Calorimetry test (1934)

The moisture content of the standardised brownie was found to be 22 % respectively. It refers to the number of water molecules that become incorporated into a food product. The Ash content of the standardised brownie were found to be 0.7 g respectively. It indicates the amount of essential minerals and trace elements present in the food product. The protein content of the standardised brownie were found to be 6.5 g respectively. The iron content of the standardised brownie were found to be 6.3 mg respectively.

Conclusion:

Today's health-conscious consumers are driving the demand for healthier bakery options. Producers are responding with innovative snacks packed with beneficial nutrients like iron and protein. Rice Flakes with Pomegranate Brownies are a prime example, offering a delicious and nutritious alternative to traditional brownies. Rich in iron and protein, these brownies are a perfect choice for those seeking a healthier treat.

References:

1. Buchowski, M. S., de la Fuente, F. A., Flakoll, P. J., Chen, K. Y., & Turner, E. A. (2001). Increased bone turnover is associated with protein and energy metabolism in adolescents with sickle cell anemia. *American Journal of Physiology-Endocrinology and Metabolism, 280*(3), E518-E527.
2. Danesi, F., & Ferguson, L. R. (2017). Could pomegranate juice help in the control of inflammatory diseases?. *Nutrients, 9*(9), 958.
3. Tsaras, G., Owusu-Ansah, A., Boateng, F. O., & Amoateng-Adjepong, Y. (2009). Complications associated with sickle cell trait: a brief narrative review. *The American journal of medicine*, *122*(6), 507-512.
4. Kanter, J., & Kruse-Jarres, R. (2013). Management of sickle cell disease from childhood through adulthood. *Blood reviews*, *27*(6), 279-287.

5. Mandese, V., Marotti, F., Bedetti, L., Bigi, E., Palazzi, G., &Iughetti, L. (2015). Effects of nutritional intake on disease severity in children with sickle cell disease. *Nutrition journal*, *15*, 1-6.
6. Obeagu, E. I., &Obeagu, G. U. (2024). Implications of climatic change on sickle cell anemia: A review. *Medicine*, *103*(6), e37127.
7. Ogedegbe, H. O. (2002). Sickle cell disease: an overview. *Laboratory medicine*, 33(7), 515-543.

PHYTOPLANKTONS OF DANDI CREEK- WEST COAST OF INDIA

Surendra S. Kadam

Department of Zoology,

Gokhale Education Society's, Arts, Commerce and Science College,

Jawhar, Dist.-Palghar, M.S. 401 701

Corresponding author Email: drsurendrk73@gmail.com

Introduction:

Diversity of phytoplankton and water quality of Dandi creek was studied during September 2009 to September 2010.Physicochemical parameters indicated variations in depth, pH, and temperature. DO, BOD, salinity, nitrite, nitrate, ammonium nitrate, phosphate etc. A total of 59 genera of phytoplankton were observed from Dandi creek system of which 50 genera were diatoms, 7 were dinoflagellates and 2 were other algae. *Coscinodisccus* was the most abundant genus in Dandi creek. Followed by *Ditylum*, *Biddulphia*, *Bacillaria*, *Certatium*, *Nitzschia*, *Naviculla*, *Plurosigma*, *Rhizosolenia*, *Thalasiossira* and *Thalassiothrix.* Variations showed in phytoplankton pigments at Dandi creek waters phytoplankton community. Variation of chlorophyll a ranged between 0.47 and 6.87 mg/m^3 (av.3.45 mg/m^3) and phaeophytin ranged between 0.18 and 2.8 mg/m^3 (av.1.04 mg/m^3). Concentration of phytoplankton pigments in the creek was fairly high. The chlorophyll a was higher in the outer zone than the interior zone of the creek. In general, phaeophytin level was lower than the chlorophyll a suggesting that the phytoplankton were in good physiological state. Seasonal variation in phytoplankton pigments was marginal. Phytoplankton pigments showed variations in concentration during pre-monsoon, monsoon and post monsoon seasons at 5 different stations of creek.

Keywords: Phytoplankton, Dandi Creek, Water Quality, Physico-Chemical Parameters

Introduction:

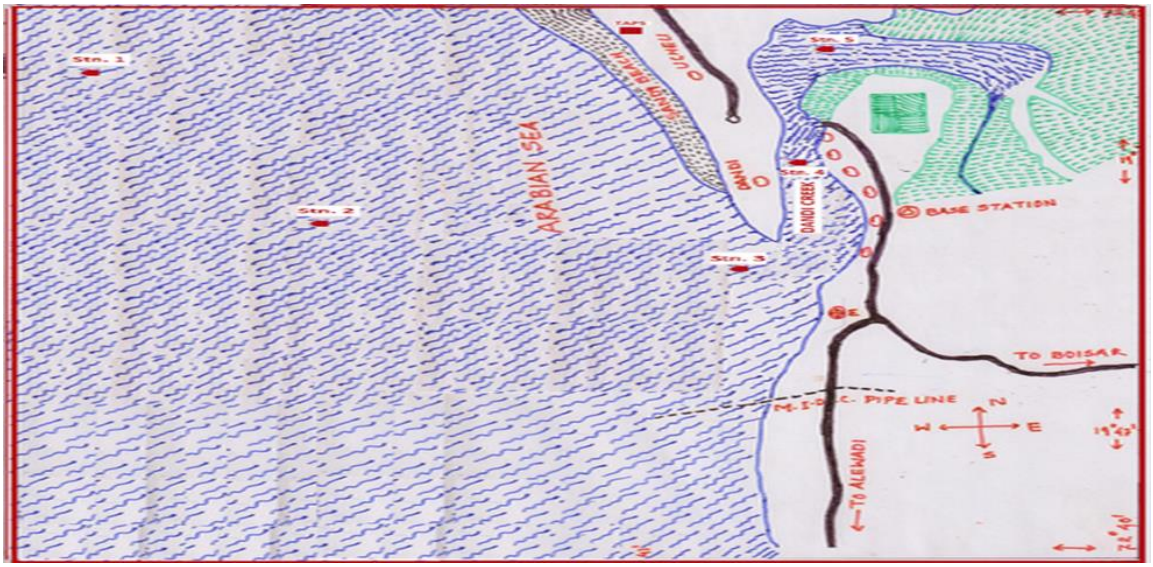
Phytoplankton communities dominate the pelagic ecosystem that cover 70% of the world's surface area. Phytoplanktons are the passively drifting plants found in the water bodies. They are autotrophic and main primary producers in the marine environment contribution about 95% of the total marine primary production, most of them are microscopic usually unicellular, solitary or chained forms. The quality and quantity of phytoplankton is good indicator of water quality (Mohammad Ali *et. al*, 2005). Marine phytoplankton which are the primary food source directly or indirectly, of all marine organisms are mainly composed of diatoms, dinoflagellates and cocolithophores, and these are major component of a marine ecosystem which initiates the

aquatic food chain. The diatoms commonly known as 'pasture of the sea' or 'meadows of the sea' are unicellular autotrophic organisms and form about 90% of phytoplankton biomass. ''The ocean's invisible forest''- marine phytoplankton play a critical role in regulating earth's climate (Falkowski, 2002).

The marine phytoplankton come in a myriad of shapes, sizes and forms, some of them quite beautiful, some drift on current while others have an ability to move around with the aid of flagella (Goswami, 2004). The present study in addition to adaptation information on ecological characteristics of phytoplankton will also help in providing baseline information for future monitoring of the system considering the industrial growth near the Dandi creek.

Study area:

The present investigation has been carried out Dandi creek of Palghar district of Maharashtra during September 2009 to September 2010; Total five station were selected for the study of Phytoplankton, out of Station 1 and 2 were located in the open sea region, station 3 was near the mouth of the creek and station 4 and 5 were within the creek area.



Material and Methods:

The water quality and phytoplankton study were carried out over a period of 13 months from September 2009 to September 2010 from 5 different locations in Dandi creek, west coast of India. Regular monthly collections were made from all the five stations. Collected samples were analyzed in the laboratory for various physico-chemical parameters, phytoplankton pigments and phytoplankton. A mechanized boat was used for the collection of samples by taking the help of local fishermen. Water samples for phytoplankton were collected from 5 different stations using clean plastic bucket and immediately transferred in 500 ml clean plastic bottles. The samples

were immediately preserved by adding 5 ml of 4 % formalin and 2ml of Lugol's iodine solution. The samples were subjected to qualitative and quantitative evaluation of phytoplankton. Quantitative evaluation was carried out by Sedgwick Rafter counting cell and for qualitative evaluation standard monographs, published research papers in national and international journals were followed. Identification of phytoplankton was doneas per Subramanyan (1959)[3]. Diversity index of phytoplankton for each month was calculated by the following formula:

Diversity index = $H = \frac{S-1}{In\ N}$

Where,

S = Number of genera of phytoplankton; N = Total number of phytoplankton

ln = Natural logarithm

Phytoplankton pigments (Chlorophyll a andPhaeophytin)

Estimation of Chlorophyll a and Phaeophytinwas carried out by Strickland and Parson Method (1972)[4]. The samples were filtered through a nylon cloth (0.3 mm mesh) followed by membrane filter (0.45 µm pore size), after adding 3 drops of 1% magnesium carbonate. The separated phytoplanktons were extracted with 90% acetone. Chlorophyll a and Phaeophytin were extracted by centrifugation and estimated spectrophotometrically by determining optical density and 665 nm before and after acidification.

Nutrients

All nutrients including Nitrate, Nitrite, Phosphate, Ammonium nitrate were analyzed calorimetrically using UV-Vis Spectrophotometer. At the time of sampling water temperature was measured by good quality thermometer. pH of samples was determined by calibrated digital pH meter. DO was measured by Winkler's Iodometric method. For the BOD direct unseeded method was employed. The sample was filled in BOD bottle in the field and was incubated in the laboratory at room temperature for 5 days after which DO was again determined, and salinity wasmeasured by Argentometric method.

Results and Discussion:

A review of various physico-chemical parameters at different locations in Dandi creek revealed that abiotic features of Dandi creek were mainly influenced by tides and monsoon of the tropics some of the parameters showed spatial variations (Table 1). Temporal and spatial variations were observed in various parameters during the present study. The creek was sea water dominated throughout the study period. Temperature of water showed marginal variation mainly influenced by atmospheric temperature. The pH of sea water was mainly alkaline except

monsoon season where it was below to standard pH value of coastal water. Salinity showed variation in premonsoon, monsoon and postmonsoon seasons.

Table 1: Variation in different physico-chemical and biological parameter of surface and bottom water

Parameters	Outer Zone		Inner Zone		Entire creek Area	
	Range	Mean	Range	Mean	Range	Mean
Temperature (^{0}C)	22.5 – 35.5	28.8	22.0 -35.5	28.7	22.0 – 35.5	29.0
pH	7.45 – 8.41	7.9	7.7 – 8.69	8.01	7.2 – 8.69	8.09
Salinity (‰)	22.23 – 38.8	30.63	19.29 – 36.34	27.93	19.29 – 38.8	30.69
DO (mg/l)	3.39 – 5.31	4.36	3.28 -5.49	4.46	3.28 – 5.49	4.40
BOD(mg/l)	0.87 – 2.12	1.50	0.78 – 2.23	1.35	0.78 – 2.23	1.32
Phosphate (µ mol/l)	0.47-3.93	1.82	0.42 -3.65	1.88	0.42-3.93	1.55
Total Phosphorus (µ mol/l)	3.41-13.6	8.21	2.3 -12.6	7.28	2.3-13.6	7.57
Nitrite (µ mol/l)	1.38- 3.78	2.59	1.38 – 3.55	2.47	1.38 – 3.78	2.52
Nitrate (µ mol/l)	23.26 - 42.28	33.39	20.93 – 39.83	29.66	20.93- 42.28	31.56
Ammonia (µ mol/l)	0.48-14.96	7.05	0.83-16.06	6.55	0.48-16.06	3.48
Total Nitrogen (µ mol/l)	41.63- 142.08	84.92	45.03- 116.63	80.14	41.63- 142.08	76.6
Chlorophyll a (mg/m3)	2.14-6.87	4.31	0.47-6.43	2.68	0.47-6.87	3.45
Phaeophytin (mg/m3)	0.5-2.66	1.43	0.18-2.08	1.14	0.18 - 2.8	1.04
Phytoplankton Cell count(no.X10^3)	284 - 3238	1645.5	88 - 2965	5.68	88 – 3238	920.28

DO & BOD showed substantial variation, the average concentration was comparable with standard values given for coastal water. DO & BOD values indicated effective assimilation of organic load. Phosphate phosphorus and total phosphorus showed slightly higher values. Nitrite nitrogen, nitrate nitrogen and total were also in the range normally found in coastal water. Ammonia nitrogen concentration was slightly higher probably due to land drainage and industrial discharge in Dandi creek.

Total 59 genera of phytoplankton were observed during the study period comprising diatoms, dinoflagellates and other algae. Phytoplankton observed from Dandi creek including *Amphiprora, Amphitetra, Amphora, Asterionella, Bacillaria, Bacteriastrum, Biddulphia, Campylodiscus, Corethron, Caloneis, Cerataulina, Chaetoceros, Climacocosphemia, Coconeis, Coscinodiscus, Diploneis, Distephanes, Ditylum, Eucampia, Fragillaria, Gramatophora, Gunardia, Gyrosigma, Hemidiscus, Isthmia, Lauderia, Leptocylindrus, Licmophora, Lyngbya, Melosira, Navicula, Nitzschia, Ornithocercus, Oxytoxum, Planktoniella, Pluerosigma, Podocystis, Rhabdonema, Rhizosolenia, Schroederella, Skeletonema, Stauroneis Stephanopyxis, Surinella, Synedra, Thalassionema, Thalassiosira, Thalassiothrix, Triceratium, Ceratium, Dinophysis, Exuviaella, Gymnodinium, Peridinium, Prorocentrum, Protoperidinium, Oscillatoria, Phaeocystis*.

Phytoplankton cell count and species diversity

The phytoplankton cell counts at station 1 ranged between 414 and 3238 X 10^3/l (av.1316.15 X 10^3/l). The highest cell count was observed in the month of March and lowest in the month of September. The average phytoplankton cell counts for premonsoon (1550 X 10^3/l), monsoon (542 X 10^3/l) and postmonsoon (2050 X 10^3/l) seasons. Diatoms constituted the major group of phytoplankton contributing 91.96% of total phytoplankton population. The percentage contribution of diatoms for this station was 91.97%. The maximum percentage contribution was observed in the month of November and the minimum January. Seasonal percentage contribution of diatoms was respectively 34.52, 15.39 and 50.10 % for premonsoon, monsoon and postmonsoon. The dinoflagellates percentage for this station was 5.92 with highest contribution in the month of March and the lowest in the month of April. Seasonal percentage contribution of dinoflagellares was respectively 61.63, 17.10 and 21.27 for premonsoon, monsoon and postmonsoon. The percentage contribution of other algae at station 1 was 2.12. Their highest contribution was observed in the month of October and the lowest in the month of March. Seasonal percentage contribution of other algae for premonsoon, monsoon and postmonsoon periods were respectively 20.56, 36.67 and 42.78.

Table 2: Variation in phytoplankton cell count (no X 10^3/l) at different stations during 2009-10

Months	Station 1	Station 2	Station 3	Station 4	Station 5
Sep,09	414	356	364	267	250
Oct	1738	1356	1268	1115	745
Nov	2381	1686	1520	1026	738
Dec	2628	2630	2205	1615	756
Jan,10	1453	1232	939	531	319
Feb	1340	995	964	576	328
Mar	3238	2563	2965	1204	661
Apr	619	425	396	299	182
May	1003	858	614	376	151
Jun	486	284	274	274	88
Jul	784	644	578	342	124
Aug	428	368	282	110	100
Sep	598	522	474	300	298

Conclusion:

Present investigation proposes that Dandi creek is fully rich with phytoplankton and supported a various community. Dandi creek an ecologically important estuarine system can be termed as slightly to moderately polluted because creek is surrounded by Tarapur industrial area and many industries are responsible for water pollution. Present baseline information on the phytoplankton pigments and community structure are useful for future ecological assessment andmonitoring of the coastal ecosystem of Dandi creek area.

Acknowledgments:

Authors are grateful to Dr.L.RTiwari, principal(Rtd.) of Maharshi Dayanand college,parel Mumbai for his constant encouragements and guidance too Dr.Anjali Kulkarni principal of N.B.Mehta Science College, Bordi for their kind support.

References:

1. Tiwari, L. R., & Nair, V. R. (1998). Ecology of phytoplankton from Dharamtar creek, west coast of India. *Indian Journal of Marine Science, 27*, 302–309.
2. Muhammad Ali *et al.* (2005). Studies on monthly variation in biological and physico-chemical parameters of Brackish water fish pond, Muzaffargarh, Pakistan. *Journal of Research (Science), Bahauddin Zakaria University, Multan, Pakistan, 16*(1), 27-38.

3. Subrahmanyan, R. (1959). Studies on the phytoplankton of the west coast of India. Pt. I. Quantitative and qualitative fluctuations in the total plankton crop, the zooplankton crop and their interrelationship, with remarks on the magnitude of the standing crop and production of matter and their relationship to fish landings. *Proceedings of the Indian Academy of Sciences, 50B*, 113-187.
4. Strikland, J. D. J., & Parsons. (1972). A practical handbook of sea water analysis (Bull. No.167). Fisheries Research Board of Canada.
5. Gonkar, C. A., Sawant, S. S., Chandrashekar, A. A., Venkat Krishnamurthy, & Gonzalves, E. A. (1947). Variations in the seasonal compositions of the phytoplankton of Bombay harbour. *Current Science, 16*, 304-305.
6. Gopinath, C. P. (1972). Seasonal abundance of phytoplankton in Cochin backwaters. *Journal of Marine Biological Association of India, 14*, 668-577.
7. Goswami, S. C. (2004). Phytoplankton Identification Manual. National Institute of Oceanography, Goa: 1-35 pp.
8. Hutchinson, G. E. (1944). Limnological studies in Connecticut III: A critical examination of the supposed relationship between phytoplankton and chemical changes in lake waters. *Ecology, 25*(1), 3-26.
9. Jayalakshmi, K. V., Kumaram, S., & Vijayan, M. (1986). Phytoplankton distribution in Cochin backwaters – Seasonal study. *Mahasagar-Bulletin of the National Institute of Oceanography, 19*(1), 29-37.
10. Kadam, A. S. (1958). Monoculture of marine planktonic diatoms and the effect of different media on their growth and survival. *Journal of Indian Fisheries Association, 19*, 69-74.
11. Kaladharan, P., Alavandi, S. V., Pillai, V. K., & Balachandran, V. K. (1990). Inhibition of primary production as induced by heavy metal ions on phytoplankton population off Cochin. *Indian Journal of Fisheries, 37*(1), 51-54.
12. Kofoid, C. A. (1903). Plankton studies IV: The plankton of Illinois River, 1894-99 with introductory notes on the hydrography of the Illinois River and its basin. Part I. Quantitative investigation and general results. *Bulletin of the Illinois Natural History Survey, 6*(2), 95-624.
13. Kotiwar, O. S. (2007). Distribution and abundance of phytoplankton along west coast of India. In *National Symposium on Conservation and Valuation of Marine Biodiversity* (pp. 201-218).
14. Lodh, N. M. (1990). Ecological studies on plankton from near shore waters of Bombay. Ph.D. Thesis, University of Mumbai, 203 pp.

A CONCISE OVERVIEW OF ANDERSEN-TAWIL SYNDROME

Ujjval P. Vaghela*[1], Bhavik Jani[2], Ishita Zalavadiya[2],
Yash Kachhadiya[2] and Hardik Radadiya[2]

[1]Department of Pharmacy,
Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
[2]School of Pharmacy, RK University, Rajkot, Gujarat, India.
*Corresponding author E-mail: ujjvalvaghela08@gmail.com

Abstract:

Mutations that disrupt the protein Kir2.1, which controls cardiac excitability, produce Andersen-Tawil syndrome (ATS), a rare hereditary illness. The KCNJ2 gene produces this protein. Atrial fibrillation syndrome (ATS) is characterized by periodic paralysis, potentially fatal ventricular arrhythmias, and dysmorphic characteristics. However, the disorder can present in many ways, and not all ATS patients have a genetic mutation. Few understand this arrhythmogenic condition's causes. Understanding these pathways helps distinguish ATS from other channelopathies with similar symptoms and develops customized treatments. Recently proposed Kir2.1 as a countercurrent to sarcoplasmic calcium reuptake may explain the arrhythmogenic processes of ATS and its overlap with catecholaminergic polymorphic ventricular tachycardia. This review summarizes the latest research on ATS arrhythmias that induce SCD. KCNJ2 loss-of-function mutations cause ATS1. This gene produces the Kir2.1 inward rectifier potassium channel. The genetic defect in ATS type 2 (ATS2) is unknown, however its symptoms are identical to ATS1. Thus, ATS2 comprises all ATS instances without a hereditary KCNJ2 mutation. Mutations in KCNJ2 affect cardiac and skeletal muscle excitability, causing heart arrhythmias and periodic paralysis of atrial fibrillation type 1 (ATS1). The molecular process behind dysmorphic characteristics is poorly understood. ATS's clinical symptoms, genetic and molecular anomalies, and treatment and management options.

Keywords: Andersen-Tawil syndrome, Protein Kir2.1, Atrial Fibrillation Syndrome, Ventricular Arrhythmias.

Introduction:

Andersen–Tawil syndrome

Ventricular arrhythmias, periodic paralysis, and bodily dysmorphologies are symptoms of the uncommon genetic condition known as Andersen-Tawil syndrome (ATS), also known as

long QT syndrome type 7 (LQT7). In 1963, a specific type of periodic paralysis associated with premature ventricular contractions was described by Klein *et al.*, who likely made the initial reference to the illness. After multiple presentations of this subtype of periodic paralysis, it wasn't until 1971 that Ellen Damgaard Andersen and colleagues provided the first comprehensive description of the condition characterized by the aforementioned trio of symptoms. However, the syndrome was not accurately documented until 1994 when Tawil and colleagues described numerous patients, purposefully using the eponym "Andersen's syndrome". It was proposed in 2003 to honor Tawil's clinical work on the disease by renaming it "Andersen-Tawil syndrome". "Andersen-Tawil syndrome" is the most commonly used term these days, while "Andersen's syndrome" or "Andersen syndrome" are equally acceptable. The autosomal recessive illness known as "Andersen disease," named after Dorothy Hansine Andersen, is caused by a lack of the glycogen branching enzyme and is distinct from the syndrome. [1,2]

The occurrence of ATS is unpredictable, and it has been found to be hereditary in an autosomal dominant manner. Even within a single ATS-affected family, the severity of symptoms can vary greatly, and there is no universal set of symptoms that all ATS patients experience. To our knowledge, ATS is caused solely by mutations in the KCNJ2 gene. Kir2 is the protein that codes for the inward rectifier potassium channel. Patients with ATS with known abnormalities in this gene are called ATS type 1 (ATS1), while patients with an unknown mutation are called ATS type 2 (ATS2). About 30 percent of ATS1 probands have a KCNJ2 mutation that is new to the gene. A seemingly de novo mutation could be the result of paternal germline mosaicism, which is a rare occurrence. This has been documented in certain cases, such as Timothy syndrome (LQT8), for instance. [3,4]

There is no known exact prevalence of ATS; however, estimates of 1/1,000 have been put up. An estimated minimum point prevalence of 0.8/1,000,000 for England in December 2011 was derived from data collected over 15 years. The real frequency of ATS is likely much greater than 1 in 100,000, given that this estimation is based on individuals sent to the UK national channelopathy program with a clinical diagnosis of periodic paralysis that could be verified with a mutation in KCNJ2. [5,6]

Signs and symptoms of cardiac problems

Tristani-Firouzi *et al.* classified ATS, or 'Andersen syndrome' as a subtype of the long QT syndrome (LQTS) due to the extensive range of cardiac symptoms observed in individuals with ATS, one of which is a lengthened QT interval. Andersen *et al.* did not include the long QT characteristic when they initially described the disease, which is something to keep in mind. The large variation in rate-corrected QT intervals (QTc intervals) seen in ATS patients, healthy

individuals, and other LQTS subtypes could account for this. While QTc intervals in individual ATS patients range from b390 ms to far beyond 500 ms, on average, they are only slightly extended. A QTc interval greater than the diagnostic cutoff of 450 ms for males and 460 ms for females is therefore not always observed in ATS patients. A pronounced U wave, as seen in Fig. 1B, which is most often seen in the anterior precordial leads of the electrocardiogram (ECG) is another cardiac symptom in individuals with ATS. The appearance of U waves at greater heart rates is indicative of illness, although they can be observed in healthy individuals at lower heart rates. According to Zhang *et al.*, out of 96 ATS1 patients from 33 different kindreds with 24 different KCNJ2 mutations, 39 patients (41% of the total) had cardiac arrhythmias on their baseline electrocardiograms.

Despite this, palpitations were reported by just 2 out of 39 patients. Syncope and/or cardiac arrest were experienced by ten patients. A total of 87 patients (91% of the total) exhibited at least one of the following T-U wave characteristics: an expanded U wave, biphasic U waves, a wide T-U junction, or a protracted terminal component of the T wave downslope. According to these trends, ATS1 genotype prediction—that is, the position of the mutation in various functional areas of the KCNJ2 gene—was quite accurate. A group of fifteen individuals with biphasic U waves served as an example for this ATS1 genotype prediction. Twelve individuals exhibited mutations in the N-terminal region of KCNJ2, whereas only one patient exhibited mutations in the M1 pore area and two patients exhibited mutations in the C-terminal region. A median QTc interval increase was seen in the patients; however, this increase was minor, with a value of 20 ms, and only 17% of patients had a QTc interval of N460 ms. In a similar vein, just three out of nineteen (16%) ATS patients were found to have a QTc interval of 460 ms or longer by Haruna *et al.* [7,8,9]

Category 1 Andersen-Tawil syndrome

The ATS1 cardiac condition is incredibly rare, with a frequency estimated at less than 1 in 100,000. It is characterized by a specific phenotype that was initially documented in 1971 by Andersen *et al.* and includes periodic paralysis, cardiac arrhythmias, and dysmorphic characteristics. In their further characterization of the condition, Tawil *et al.* There is an autosomal dominant pattern of inheritance for KCNJ2 mutations that produce ATS1. However, de novo mutations occur at a high pace during embryonic development. Approximately 35% of the mutations were found to be new in a recent study that included 118 patients with ATS1 from 57 different families. The association between ATS1 and certain KCNJ2 gene variants was initially documented by Plaster *et al.* The dominant-negative loss-of-function of Kir2.1 channels

is the consequence of most ATS1 mutations. This has a direct impact on the robust inward rectifier potassium current (IK1), which in turn leads to its significant downregulation. [10]

Trisomy 21 is characterized by several phenotypic manifestations, including but not limited to bidirectional ventricular tachycardia (BiVT), extrasystoles (typically bigeminy), muscular weakness, and sustained periodic paralysis. It is also characterized by dysmorphic features, including a wide forehead, hypoplastic mandible, hypotelorism, low-set ears, and digit clinodactyly, among many others. Between 35% and 50% of ATS1 individuals experienced recurring episodes of muscle weakness, and approximately 67% to 75% displayed dysmorphic characteristics in published reports. However, 90% of patients showed a noticeable U-wave on the electrocardiogram (ECG), and 97% of patients had at least one cardiac abnormality. Sixty to ninety percent of ATS patients have ventricular arrhythmias, with biventricular tachycardia and polymorphic ventricular tachycardia (PVT) both being very common. Since KCNJ2 mutations might vary in their expressivity, the ATS1 pleiotropic phenotype could be diverse. Patients of the same genetic line who have impaired or non-existent Kir2.1 current may exhibit a wide range of symptoms. Because some ATS1 patients may not have any symptoms at all, while others in the same family may exhibit all three diagnostic signs, in varying degrees and combinations, making a definitive diagnosis challenging.

Furthermore, for early-onset types of the disease, family history is unlikely to be of substantial assistance because some causal mutations develop de novo. At this time, the criteria that Venance and colleagues have developed are used to make diagnoses. When trying to explain the pathogenesis of ATS1, these writers mostly look at isolated periodic paralysis, polymorphic ventricular ectopy, and characteristic ATS dysmorphias. The true number of instances can be underreported due to the difficulty in diagnosing the disease. Unless otherwise stated, the following discussion will center on the electrical symptoms and potential causes of the condition as it affects the heart. electrocardiographic results specific to ATS1. These features indicate the presence of focal arrhythmias such as bigeminy, ventricular ectopy, BiVT, and PVT in the form of Torsades de Pointes. They include delayed cardiac repolarization, a prolonged T wave downslope, a wide T-U junction with a high amplitude, and broad U waves. This potentially fatal arrhythmia can resolve on its own or progress to ventricular fibrillation (VF), which in turn can cause cardiac arrest (CA) accompanied by syncope or sudden cardiac death (SCD). Although Delannoy *et al.* observed 2.3% SCD in ATS1 patients, Mazzanti *et al.*1 reported 9.3%. Among younger people, SCD poses the greatest threat. Some patients show signs of conduction abnormalities, including non-specific intraventricular conduction delay, right or left bundle branch block, and first-degree atrioventricular (AV) block. We are unaware of any other cases of

total AV block in ATS patients; hence, doctors may need to take this into account as a potential associated symptom when deciding on the best course of treatment. The catecholaminergic polymorphic ventricular tachycardia (CPVT) condition, which is primarily linked to polymorphisms in the human cardiac Ryanodine Receptor 2 (RyR2) gene that encodes the cardiac RyR2 channel, overlaps with ATS in approximately 40% of patients.

Considering Kir2.1's potential function as a countercurrent to sarcoplasmic calcium reuptake and its recently noted functional expression in the sarcoplasmic reticulum (SR), this overlap could be explained. While ATS1 was first identified as a member of the congenital long QT syndrome (LQTS) family of channelopathies, namely LQT type 7, the QT prolongation is now more associated with a strong U-wave than with an inherent corrected QT (QTc) prolongation. The fact that QT prolongation accounts for just 15% of the phenotypic manifestation also casts doubt on the hypothesis that ATS1 is a member of the LQTS family.1 Although there is minimal evidence linking KCNJ2 to LQTS, a recent study by Alder *et al.* demonstrated that just three out of seventeen (18%) genes associated with LQTS have been definitively connected to typical LQTS. It is more accurate to classify ATS1 as an independent hereditary channelopathy rather than a subtype of LQTS because most ATS1 patients do not exhibit QT interval prolongation and the mutations cause a variety of cardiac and muscle-skeletal abnormalities. [11,12,13]

The ATS1 arrhythmogenesis source

Kir2.1's encoding gene, KCNJ2, is a part of subfamily J of inwardly rectifying potassium channels, which also comprise KCNJ12, KCNJ4, KCNJ14, KCNJ17, and KCNJ18, which code for Kir2.2, Kir2.3, Kir2.4, Kir2.5, and Kir2.6, respectively. Neurons, pulmonary smooth muscle cells, and aortic endothelial cells express Kir2.4, whereas skeletal muscle is the only organ to express Kir2.6. The other four Kir2.x isoforms are extensively expressed in the heart of mammals, and they are also present in the brain, skeletal muscle, and vascular muscle. As with all Kir channels, Kir2.1 has two M1 and M2 transmembrane helix domains, a loop that forms pores selectively for ions between M1 and M2, and amino (NH2) and carboxyl (COOH) terminals in the cytoplasm. Characteristically distributed differentially across the atrium and the ventricles, the subunits that make up functional Kir2.x channels co-assemble in either a homo- or heterotetrameric fashion. The inwardly rectifying channels in the ventricles are mostly formed by Kir2.1, while Kir2.3 is mostly found in the atria.

In humans, Kir2.2 and Kir2.1 do not coexist. The functional characteristics of IK1, the inwardly rectifying potassium current that these channels conduct, vary with the stoichiometry of the monomers that make up the tetramer, hence this is vital to know.34 Furthermore, the

syndromic phenotype should be modified by the configuration of individual Kir2.x channel tetramers, whose unique features should determine the implications of ATS1-associated mutations. The pleiotropy observed in ATS1 individuals can be partially explained by the variable composition of the channels. On a functional level, IK1's current-voltage (I/V) relationship in the atrium and ventricles exhibits a reversal potential near the resting membrane potential and a negative slope conductance at depolarized potentials ranging from -50 to 0 mV. So, although IK1 is the most conductance at the RMP, it drops to zero when the action potential (AP) plateau is reached.35 Thus, this non-linear I/V relationship in the heart shows how IK1 aids in controlling the RMP, depolarization toward threshold, plateau shape and duration, and eventual phase of AP repolarization.

When the cell membrane becomes too polarized, intracellular magnesium ions and polyamines bind to negatively charged residues inside the cytoplasmic pore of the Kir2.x channel, blocking the outward flow of potassium ions and causing the channel to re-form. According to Kir2.1 channels have various locations for binding Mg2+ and polyamines, with the most concentration at D172, E224, and E299 charges. A channel's inwardly rectifying response becomes faulty when any of these residues undergo mutation in IK1. Particularly, Kir2.1 gain-of-function mutations leading to short QT syndrome include amino acid substitutions at residues D172 and E299, identified as D172N and E299V, respectively. Due to both alterations, the QT interval on electrocardiograms and the action potential duration (APD) in individual cells are both shortened. Similarly, it has been demonstrated that class 1c antiarrhythmic medications, such as propafenone and flecainide, bind to the Kir2.1 channel's Cysteine 311 (Cys311) residue, decreasing the polyamine-induced inward rectification and increasing outward IK1. 43,44 dollars Therefore, ATS1 patients often consider using propafenone/flecainide either alone or in combination with β-blockers, which stop the arrhythmic effects of adrenaline. [14,15]

There are additional systems that control Kir2.1 function. The cytoplasmic leaflet of Kir2.x channels is coupled to a membrane-anchored phospholipid called phosphatidylinositol 4,5-bisphosphate, or PIP2, which acts as a second messenger to control channel gating. PIP2 has a competitive role in inhibiting Kir2.1 channel activity, whereas other PIPs have the reverse effect. However, channel activity and protein-protein interaction are made possible through phosphorylation of Kir2.1 channels, which is mostly mediated by protein kinase pathways, including PKA and PKC, respectively. Total channel inactivation occurs as a consequence of adrenergically driven phosphorylation of PKA. Arrhythmogenesis occurred in Purkinje myocytes of dogs, guinea pigs, and mice when isoproterenol-induced PKA activation blocked Kir2.1

function. Hence, it should be emphasized that adrenergically induced IK1 depletion can impact AP repolarization, resulting in early afterdepolarizations and activated activation. [16,17]

Therapeutic mechanism

There is currently no effective therapy available for ATS sufferers, as seen by the variety of therapeutic possibilities they have. Also, the decision to undertake catheter ablation is based on data that suggests the left ventricular Purkinje fibers could be a focal cause of ventricular arrhythmias in ATS patients. Radiofrequency catheter ablation has been tried and failed in every case where it has been recommended as a treatment. This is probably due to the fact that the genetic abnormality and phenotypic cannot be pinpointed to a specific location. There are currently no set clinical guidelines for atrial fibrillation (ATS), and the only agreed-upon approach is to avoid prescribing drugs that are known to increase the QT interval to individuals with this condition.

To this day, pharmacologic treatment for arrhythmias in ATS1 is still symptom-based and lacks specificity. It relies on amiodarone and class Ic antiarrhythmic medications (such as flecainide and propafenone) to lessen the likelihood of arrhythmias. Flecainide is currently the most popular treatment for ATS patients, despite its low efficacy rate, and this is without taking into account any underlying processes. The cytoplasmic domain of Kir2 is bound by both flecainide and propafenone. Along with lowering NaV1.5 channel function, they decrease Likewise, at clinically relevant concentrations, both medications block the KCNH2-encoded 'hERG' potassium channels that are responsible for the rapid delayed rectifier potassium current (IKr). This underscores the importance of closely monitoring patients with other risk factors for acquired LQTS or not giving them the drugs at all. It is recommended to avoid amiodarone due to its increased risk of cardiac events, and according to Mazzanti *et al.*, these medicines rarely work together to decrease arrhythmias in ATS1. Although the danger of sudden cardiac death (SCD) is still present, implantable cardiac defibrillators are often required in cases of severe and symptomatic cardiac arrhythmias.one thousand twelve hundred Since the underlying mechanisms are not well known, proarrhythmia frequently occurs as a consequence of treatment that is empirical and based on clinical judgment. Consequently, ATS1 must be further understood and treated immediately. In the years to come, medication design should still be relevant. The identification of an effective pharmacophore relies heavily on the chemical characterisation of targets of potential activating or inhibitory compounds. To better categorize and predict the fate of people afflicted by disorders like ATS1, research into the efficacy of currently authorized medications and their derivatives should persist. Conversely, new insights into the various arrhythmogenic mechanisms linked to ATS1 mutations may pave the door for

more targeted therapies for individuals afflicted with this disastrous channelopathy. Each patient should receive individualized pharmacological treatment and clinical management for ATS1 due to the fact that the molecular pathways causing the disease vary with each mutation. Take ATS1 patients who carry the Kir2.1 mutation as an example.

This mutation impacts both Kir2.1 and NaV1.5 membrane trafficking, which means that their cardiac excitability will likely be significantly reduced. Consequently, treatment with sodium channel blockers such as flecainide or propafenone should be avoided due to the possibility of proarrhythmia. It is worth considering compounds that can optimize non-conventional transport pathways of trafficking-deficient mutations as a means to reduce pathogenicity in these patients. On the flip side, patients with mutations that impact the interactions between Kir2.1 and PIP2 but do not impact sodium channel function may get relief from existing antiarrhythmics. Put simply, a patient with a type A ATS1 mutation that causes defects in Kir2.1 trafficking might be put at risk by using a specific combination of β-blockers and other antiarrhythmics, even though this combination might be suitable for a patient with a type B ATS1 mutation that changes Kir2.1 channel conformation. Thus, it is important to tailor medication treatment for ATS1 mutations. [18,19,20]

Conclusion:

ATS is an uncommon genetic multisystem illness about 1:100,000. However, it is a fascinating "model disease" that has been the subject of many clinical and preclinical studies. This review covers ATS symptoms, their molecular and genetic causes, and how to best treat the syndrome's disorders. The ATS phenotype includes cardiac arrhythmias, periodic paralysis, and dysmorphologies, however not all patients have them. Even within a family, symptoms vary in severity. ATS's cardiac phenotype includes a prolonged QT interval, a pronounced U wave, especially on the precordial leads, and ventricular arrhythmias of varied severity, likely caused by calcium excess in a destabilized resting membrane potential. Emerging evidence links ATS to neurocognition. Despite advances in understanding and technology, much about ATS's molecular origins and genetics remains unclear. Future studies should focus on the genetic causes of ATS type 2 and how ATS mutations produce dysmorphologies.

References:

1. Andersen, E. D., Krasilnikoff, P. A., & Overvad, H. (1971). Intermittent muscular weakness, extrasystoles, and multiple developmental abnormalities: a new syndrome? Acta Paediatrica Scandinavica, 60, 559–564.

2. Tawil, R., Ptacek, L. J., Pavlakis, S. G., *et al.* (1994). Andersen's syndrome: potassium-sensitive periodic paralysis, ventricular ectopy, and dysmorphic features. Annals of Neurology, 35, 326–330.
3. Sansone, V., Griggs, R. C., Meola, G., Ptacek, L. J., Barohn, R., Iannaccone, S., Bryan, W., Baker, N., Janas, S. J., Scott, W., Ririe, D., & Tawil, R. (1997). Andersen's syndrome: a distinct periodic paralysis. Annals of Neurology, 42, 305–312.
4. Canun, S., Perez, N., & Beirana, L. G. (1999). Andersen syndrome autosomal dominant in three generations. American Journal of Medical Genetics, 85, 147–156.
5. Plaster, N. M., Tawil, R., Tristani-Firouzi, M., *et al.* (2001). Mutations in Kir2.1 cause the developmental and episodic electrical phenotypes of Andersen's syndrome. Cell, 105, 511–519.
6. Tristani-Firouzi, M., Jensen, J. L., Donaldson, M. R., *et al.* (2002). Functional and clinical characterization of KCNJ2 mutations associated with LQT7 (Andersen syndrome). Journal of Clinical Investigation, 110, 381–388.
7. Tester, D. J., Arya, P., Will, M., *et al.* (2006). Genotypic heterogeneity and phenotypic mimicry among unrelated patients referred for catecholaminergic polymorphic ventricular tachycardia genetic testing. Heart Rhythm, 3, 800–805.
8. Andelfinger, G., Tapper, A. R., Welch, R. C., Vanoye, C. G., George, A. L. Jr., & Benson, D. W. (2002). KCNJ2 mutation results in Andersen syndrome with sex-specific cardiac and skeletal muscle phenotypes. American Journal of Human Genetics, 71, 663–668.
9. Davies, N. P., Imbrici, P., Fialho, D., *et al.* (2005). Andersen-Tawil syndrome: new potassium channel mutations and possible phenotypic variation. Neurology, 65, 1083–1089.
10. Khan, I. A. (2002). Long QT syndrome: diagnosis and management. American Heart Journal, 143, 7–14.
11. Zhang, L., Benson, D. W., Tristani-Firouzi, M., *et al.* (2005). Electrocardiographic features in Andersen-Tawil syndrome patients with KCNJ2 mutations: characteristic T-U-wave patterns predict the KCNJ2 genotype. Circulation, 111, 2720–2726.
12. Tsuboi, M., & Antzelevitch, C. (2006). Cellular basis for electrocardiographic and arrhythmic manifestations of Andersen-Tawil syndrome (LQT7). Heart Rhythm, 3, 328–335.
13. Schoonderwoerd, B. A., Wiesfeld, A. C., Wilde, A. A., *et al.* (2006). A family with Andersen-Tawil syndrome and dilated cardiomyopathy. Heart Rhythm, 3, 1346–1350.

14. McManis, P. G., Lambert, E. H., & Daube, J. R. (1986). The exercise test in periodic paralysis. Muscle & Nerve, 9, 704–710.
15. Fournier, E., Arzel, M., Sternberg, D., *et al.* (2004). Electromyography guides toward subgroups of mutations in muscle channelopathies. Annals of Neurology, 56, 650–661.
16. Katz, J. S., Wolfe, G. I., Iannaccone, S., Bryan, W. W., & Barohn, R. J. (1999). The exercise test in Andersen syndrome. Archives of Neurology, 56, 352–356.
17. Jongsma, H. J., & Wilders, R. (2001). Channelopathies: Kir2.1 mutations jeopardize many cell functions. Current Biology, 11, R747–750.
18. Lopatin, A. N., & Nicholas, C. G. (2001). Inward rectifiers in the heart: an update on Ik1. Journal of Molecular and Cellular Cardiology, 1, 625–638.
19. Chen, L., Kawano, T., Bajic, S., *et al.* (2002). A glutamate residue at the C-terminus regulates activity of inward rectifier K channels: implication for Andersen's syndrome. Proceedings of the National Academy of Sciences USA, 99, 8430–8435.
20. Hosaka, Y., Hanawa, H., Washizuka, T., *et al.* (2003). Function, subcellular localization and assembly of a novel mutation of KCNJ2 in Andersen's syndrome. Journal of Molecular and Cellular Cardiology, 35, 409–415.

A COMPREHENSIVE REVIEW OF ASHERMAN'S SYNDROME

Bhavik Jani*[1], Ishita Zalavadiya[1], Nathabhai Babriya[2],
Bhavesh Thakkar[1] and Shaikh Mohammad Akil[1]
[1]School of Pharmacy, RK University, Rajkot, Gujarat, India
[2]N. R. Vekariya institute of Pharmacy- Junagadh, Gujarat, India
*Corresponding author E-mail: bhavik.jani92@gmail.com

Abstract:

Asherman's Syndrome, also known as intrauterine adhesions or synechiae, is a rare but clinically significant condition characterized by the formation of fibrous tissue within the uterine cavity, resulting in adhesions and obliteration of the endometrial cavity. This condition often leads to a spectrum of menstrual disturbances, infertility, and recurrent pregnancy loss. The purpose of this comprehensive review article is to elucidate the multifaceted aspects of Asherman's Syndrome, encompassing its pathophysiological mechanisms, diagnostic challenges, and various treatment modalities. A thorough understanding of this syndrome is crucial for healthcare providers to offer optimal care and management options to affected patients, ultimately improving their reproductive outcomes and quality of life.

Keywords: Asherman's Syndrome, Intrauterine Adhesions, Pathophysiology, Treatment

Introduction:

Heinrich Fritsch first described a case of posttraumatic intrauterine adhesion. In 1950 Joseph G. Asherman described the history of 29 women with amenorrhea secondary to trauma of the uterine cavity, the Asherman's syndrome (AS). He described in detail the etiology, anatomy, differential diagnosis, and prognosis of this disease. Later, in 1950, he also described a treatment by laparotomy and the use of intrauterine catheters to prevent its recurrence. AS is defined by the presence of intrauterine adhesions or adhesions in the endocervix with consequent risk of hypomenorrhea/amenorrhea, reduced fertility, pregnancy loss and abnormal placentation. [1] The recently pregnant uterus seems susceptible to trauma of the basal layer of the endometrium, which can develop into intrauterine adhesions and may give future problems for the patient. It is estimated that more than 90% of cases with AS occur after pregnancy-related curettage. There have been an increasing number of cases of this syndrome described worldwide. From Asherman's original definition, the syndrome was a consequence of trauma to the endometrium, producing partial or complete obliteration in the uterine cavity and/or the cervical canal, resulting

in conditions such as menstrual abnormalities, infertility, and recurrent pregnancy loss. The term ‘‘syndrome’’ in Greek refers to ‘‘concurrence of symptoms,’’ a group of symptoms that collectively indicate or characterize a disease.

In this respect, the diagnosis of Asherman syndrome should be made only in women with clinical symptoms. Adhesions are composed of fibrotic tissue, which may result in the adherence of opposing surfaces. It is possible that, after injury to the endometrium, fibrosis may follow with the potential for adhesion formation. The original description of Asherman syndrome referred to intrauterine adhesions, not fibrosis. In cases where intrauterine adhesions (IUA) are found accidentally without any symptoms one should avoid the term AS and instead apply the term “asymptomatic intrauterine adhesions.” Intrauterine adhesions can be either primary after pregnancy-related curettage, alternatively after hysteroscopic surgery, or secondary when re-occurring after adhesiolysis has been performed. It is impossible to detect or estimate the true prevalence of all IUA, as probably most cases are without symptoms. Only cases with AS, which imply pain, bleeding disorders or impaired fertility needs treatment. [2,3] Endometrial injury (especially of the gravid uterus) leads up to the development of AS as damage to the basal layer of the endometrium results in its subsequent replacement with the fibrous tissue generating intrauterine adhesions. Even in cases where pregnancy is achieved, Asherman's Syndrome can lead to a host of obstetric complications, including preterm birth, placental abnormalities, and an increased risk of cesarean section. The most common causes contributing to the development of AS include curettage of the postpartum or post abortus uterus, some infections, uterine surgeries, and the utilization of compressive sutures for postpartum hemorrhage. [4,5]

Etiology

Trauma to a gravid uterine cavity is known to be the main cause of Asherman syndrome. Such trauma could be induced by uterine curettage in the postpartum period after spontaneous miscarriage or during termination of pregnancy, or by cesarean section. One of the possible explanations for the gravid uterus being a major predisposing factor to Asherman syndrome is the low estrogen status at the time of the operation or immediately afterward, as the endometrium depends on estrogen for regeneration. Another possible explanation could be the physiologic changes that occur in a gravid uterus around the pregnancy period. [6] Trauma to a nongravid uterine cavity could also result in Asherman syndrome. The development of intrauterine adhesions can also result from various forms of hysteroscopic surgery. There has been some controversy over the exact mechanism whereby infection could result in Asherman syndrome. Several investigators maintained that the primary cause of intrauterine adhesions has been infection, especially chronic or subacute endometritis. Endometritis is unlikely to be a major

factor in the pathogenesis of intrauterine or endocervical adhesion. Endometrial tuberculosis may result in severe intrauterine adhesions, often with total obliteration of the uterine cavity and total destruction of the endometrium with resulting amenorrhea and infertility. In addition to the above predisposing factors, there may be an individual constitutional element, as some patients have apparently developed a severe form of intrauterine adhesions after a gentle suction curettage procedure, or for no apparent reason. [7,8]

Epidemiology

Asherman's Syndrome, also known as intrauterine adhesions or synechiae, is a relatively rare yet clinically significant gynecological condition that warrants detailed epidemiological scrutiny. This enigmatic disorder is characterized by the development of fibrous intrauterine adhesions, which can lead to partial or complete obliteration of the uterine cavity, resulting in a myriad of menstrual disturbances, infertility, and recurrent pregnancy loss. [9,10] The incidence varies widely across different clinical settings, ranging from approximately 2% to 30%, depending on the patient population and the specific risk factors present. [11]

Pathophysiology

Asherman syndrome is a condition in which the endometrium becomes fibrosed. The endometrial stroma is largely replaced by fibrous tissue, and the glands are usually represented by an inactive cubo-columnar epithelium of the endometrial type. The distinction between the functional and basal layer of the endometrium is lost, and the functional layer is replaced by an epithelial monolayer, which is nonresponsive to hormone stimulation and fibrous synechiae forms across the cavity. Adhesions may involve different layers of the endometrium, myometrium, or connective tissue. Adhesions derived from each of these tissues exhibit a characteristic hysteroscopic picture: endometrial adhesions are quite similar in appearance compared with the surrounding endometrium.

Myofibrous adhesions, which are most often encountered, are characterized by the presence of a thin layer of overlying endometrium, the surface of which is furnished with many glandular ostia. The surface of connective tissue adhesions lacks an endometrial lining and contrast markedly with the adjacent endometrium. Fibrous adhesions that show dense connective tissue exhibit no lining in contrast to surrounding endometrium. The Asherman's Syndrome involves a cascade of events, beginning with uterine trauma, most commonly associated with dilation and curettage (D&C) procedures following miscarriage, elective abortion, or postpartum complications. [12] Although a severe degree of intrauterine adhesions causes obstruction of the cavity and infertility, a milder degree of adhesions may be associated with repeated pregnancy loss.

Treatments

Women who have undergone uterine surgeries, such as dilation and curettage (D&C), hysteroscopic procedures, or myomectomy, may develop Asherman's Syndrome as a sequela. Additionally, individuals undergoing fertility treatments, such as in vitro fertilization (IVF), may experience treatment failure or suboptimal outcomes due to the presence of undiagnosed intrauterine adhesions. Asherman's Syndrome exhibits a broad spectrum of clinical manifestations, ranging from subtle menstrual abnormalities to profound reproductive challenges. [13,14] Timely recognition of these clinical signs and symptoms, combined with appropriate diagnostic evaluation, is pivotal for delivering optimal care to affected individuals. A comprehensive understanding of the clinical spectrum of Asherman's Syndrome is indispensable for healthcare providers, facilitating early intervention, and improving the overall reproductive outcomes and quality of life for those grappling with this condition.

Clinical assessment

Clinical evaluation serves as the initial step in diagnosing Asherman's Syndrome. Physicians should gather a detailed medical history, paying particular attention to any uterine surgeries, obstetric events, or gynecological procedures that the patient may have undergone. Menstrual history, including changes in menstrual flow and the presence of dysmenorrhea, should be thoroughly documented. Additionally, a history of infertility, recurrent pregnancy loss, or obstetric complications can be suggestive of the condition. [15]

Hysterosalpingography (HSG)

Historically, the diagnosis of AS was primarily made using radiographic techniques such as Hysterosalpingogram (HSG). Although HSG is an invasive investigation, its role is of particular importance in cases of infertility associated with AS because of its ability to demonstrate tubal patency as well as tubal contour. The drawback of HSG is that it is not useful in cases of lower uterine cavity adhesions, given the inability to fill the endometrial cavity with contrast solution. The sensitivity of HSG is 75% in the detection of IUAs while its positive predictive value (PPV) is 50%.

Ultrasound Ultrasonography (USG)

It is a non-invasive diagnostic investigation that is commonly used as the first-line diagnostic imaging modality in symptomatic patients with gynecologic pathology. IUAs appear as dense echoes within the endometrial cavity with an irregular, thin endometrium. USG is especially useful in women with dense lower uterine cavity adhesions where the role of HSG is limited. The sensitivity and specificity of transvaginal USG are 52% and 11%, respectively. Currently, 3D USG is also used to detect IUAs with a specificity of 45%.

Sonohysterography- Sonohysterography (SHG) combines the advantages of both HSG and USG. In this procedure, saline solution is instilled into the uterus to distend the endometrial cavity. The visualization of echogenic areas within the saline-filled cavity denotes the presence of IUAs. The sensitivity of SHG has been reported to be 75% and PPV of 42.9%, which is comparable to that of HSG. [16,17]

MRI- Magnetic resonance imaging (MRI)

It is another noninvasive imaging modality that can be used in cases of suspected AS. It is frequently used in cases of dense adhesions as it can evaluate the uterine cavity above the adhesions and thus helps in the evaluation of post-treatment prognosis of the disease. It is also useful in cases where the diagnosis of AS is difficult using HSG and USG due to dense adhesions and lower uterine cavity obliteration. Low-intensity signals on T2 weighted images reveal the presence of IUAs on MRI. However, its role in routine clinical practice remains limited because of its high cost. [18]

Hysteroscopy

Hysteroscopy is currently the gold standard diagnostic and therapeutic modality for the diagnosis of AS, generating only minimal patient discomfort, and allowing the accurate diagnosis and treatment in only one intervention in an office setting in selected cases, thus improving patient compliance and cost-effectiveness.

Adhesion barrier agents

To prevent the reformation of adhesions following hysteroscopic adhesiolysis, adhesion barrier agents such as hyaluronic acid gel or autocrosslinked hyaluronic acid gel can be introduced into the uterine cavity. These agents act as a physical barrier, reducing the risk of adhesion recurrence by separating the uterine walls during the postoperative healing period. [19]

Estrogen therapy

Estrogen therapy, often administered after adhesiolysis, is utilized to promote endometrial regeneration and healing. Oral, transdermal, or intravaginal estrogen supplementation is commonly prescribed to stimulate the growth of a functional endometrial layer.

Intrauterine Devices (IUDS)

In some cases, intrauterine devices (IUDs) coated with copper or levonorgestrel may be employed to prevent recurrent adhesions and maintain uterine cavity integrity. IUDs can serve as both a contraceptive method and a therapeutic strategy to minimize the risk of adhesion recurrence. [20]

Hormone therapy

In some instances, hormonal therapies may be prescribed to support endometrial growth and prevent reformation of adhesions. Hormones like estrogen and progesterone are used in a controlled manner to promote a healthy uterine environment.

Serial Hysteroscopy and Monotoring

Patients with severe Asherman's Syndrome may require multiple hysteroscopic procedures, performed at intervals, to address residual adhesions and monitor the progression of uterine healing. Serial hysteroscopy allows for a proactive approach in managing adhesion recurrence and ensuring optimal uterine function. [21]

Future perspectives:

Adult stem cells (SCs), also referred to as tissue-specific SCs, play importantroles in tissue repair and reconstruction. They proliferate by asymmetric cell division, eventually differentiating into specific cell lineages. It had long been speculated that endometrial SCs existed, based on the fact that endometrium is a dynamic tissue regenerating in every menstrual cycle. In 2004, adult SCs were first isolated from the endometrium. Three kinds of SC exist in the human endometrium: epithelial, mesenchymal and endothelial SCs. Mounting evidence has confirmed that there are SCs in both the functionalis and basalis of the human endometrium. [22] As endometrial SCs have a key role in maintaining tissue homeostasis, it is likely that their function is aberrant in disorders associated with inadequate endometrial proliferation. Specifically, in AS, it is hypothesized that there is a loss of endometrial SCs, which may or may not be dysfunctional. Therefore, SCs therapy holds great promise for the treatment of this disorder. Bone marrow-derived stem cells (BMDSCs) have also been explored as a new therapeutic strategy in AS.

Conclusion:

In the realm of gynecological disorders, Asherman's Syndrome, also known as intrauterine adhesions or synechiae, stands as a formidable and multifaceted challenge. This intricate condition, characterized by the formation of fibrous tissue within the uterine cavity, has a profound impact on the reproductive health and overall quality of life of affected individuals. Our journey through the exploration of Asherman's Syndrome has revealed a compelling narrative of pathophysiological intricacies, diagnostic intricacies, and evolving therapeutic paradigms.

References:

1. Fritsch, H. (1894). Ein fall von volligen Schwund Der Gebärmutterhohle nACh Auskratzung. Zentralblatt für Gynaekologie, 18, 1337–1342.

2. Asherman, J. G. (1950). Traumatic intra-uterine adhesions. Journal of Obstetrics and Gynaecology of the British Empire, 57(6), 892–896.
3. Schenker, J. G., & Margalioth, E. J. (1982). Intrauterine adhesions: An updated appraisal. Fertility and Sterility, 37(5), 595–610.
4. Hanstede, M. M. F., van der Meij, E., Goedemans, L., & Emanuel, M. H. (2015). Results of centralized Asherman surgery, 2003–2013. Fertility and Sterility, 104(6), 1561–1568.
5. Yu, D., Wong, Y. M., Cheong, Y., Xia, E., & Li, T. C. (2008). Asherman syndrome – One century later. Fertility and Sterility, 89(4), 759–779.
6. Diamond, M. P., & Freeman, M. L. (2001). Clinical implications of postsurgical adhesions. Human Reproduction Update, 7(6), 567–576.
7. Hellebrekers, B. W., Trimbos-Kemper, T. C., Trimbos, J. B., Emeis, J. J., & Kooistra, T. (2000). Use of fibrinolytic agents in the prevention of postoperative adhesion formation. Fertility and Sterility, 74(2), 203–212.
8. Baradwan, S., Baradwan, A., & Al-Jaroudi, D. (2018). The association between menstrual cycle pattern and hysteroscopic March classification with endometrial thickness among infertile women with Asherman syndrome. Medicine, 97(27), e11314.
9. Westendorp, I. C., Ankum, W. M., Mol, B. W., & Vonk, J. (1998). Prevalence of Asherman's syndrome after secondary removal of placental remnants or a repeat curettage for incomplete abortion. Human Reproduction, 13(12), 3347–3350.
10. Salzani, A., Yela, D. A., Gabiatti, J. R. E., Bedone, A. J., & Monteiro, I. M. U. (2007). Prevalence of uterine synechia after abortion evacuation curettage. Sao Paulo Medical Journal, 125(5), 261–264.
11. March, C. M., Israel, R., & March, A. D. (1978). Hysteroscopic management of intrauterine adhesions. American Journal of Obstetrics and Gynecology, 130(6), 653–657.
12. Hooker, A. B., Lemmers, M., Thurkow, A. L., *et al.* (2014). Systematic review and meta-analysis of intrauterine adhesions after miscarriage: Prevalence, risk factors and long-term reproductive outcome. Human Reproduction Update, 20(2), 262–278.
13. Kjer, J. J. (2014). Asherman syndrome in a Danish population. Acta Obstetricia et Gynecologica Scandinavica, 93(4), 425–427.
14. Chen, L., Zhang, H., Wang, Q., *et al.* (2017). Reproductive outcomes in patients with intrauterine adhesions following hysteroscopic adhesiolysis: Experience from the largest women's hospital in China. Journal of Minimally Invasive Gynecology, 24(2), 299–304.

15. Tsapanos, V. S., Stathopoulou, L. P., Papathanassopoulou, V. S., & Tzingounis, V. A. (2002). The role of Seprafilm bioresorbable membrane in the prevention and therapy of endometrial synechiae. Journal of Biomedical Materials Research, 63(1), 10–14.
16. Hooker, A. B., de Leeuw, R., van de Ven, P. M., *et al.* (2017). Prevalence of intrauterine adhesions after the application of hyaluronic acid gel after dilatation and curettage in women with at least one previous curettage: Short-term outcomes of a multicenter, prospective randomized controlled trial. Fertility and Sterility, 107(5), 1223–1231.
17. Rein, D. T., Schmidt, T., Hess, A. P., Volkmer, A., Schöndorf, T., & Breidenbach, M. (2011). Hysteroscopic management of residual trophoblastic tissue is superior to ultrasound-guided curettage. Journal of Minimally Invasive Gynecology, 18(6), 774–778.
18. Early pregnancy complication and abortion database [Danish]. Available from: https://www.sundhed.dk/content/cms/67/4667_tigrab_aarsrapport_2017_offentlig.pdf. Accessed March 13, 2019.
19. Sharma, J. B., Roy, K. K., Pushparaj, M., *et al.* (2008). Genital tuberculosis: An important cause of Asherman's syndrome in India. Archives of Gynecology and Obstetrics, 277(1), 37–41.
20. Goojha, C. A., Case, A., & Pierson, R. (2010). Development of Asherman syndrome after conservative surgical management of intractable postpartum hemorrhage. Fertility and Sterility, 94(3), 1098.e1–1098.e5.
21. Tonguc, E. A., Var, T., Yilmaz, N., & Batioglu, S. (2010). Intrauterine device or estrogen treatment after hysteroscopic uterine septum resection. International Journal of Gynaecology and Obstetrics, 109(3), 226–229.
22. Papoutsis, D., Georgantzis, D., Daccò, M. D., *et al.* (2014). A rare case of Asherman's syndrome after open myomectomy: Sonographic investigations and possible underlying mechanisms. Gynecologic and Obstetric Investigation, 77(3), 194–200.

Research and Reviews in Animal Science Volume III (ISBN: 978-93-95847-28-5)

About Editors

Dr. Surendra S. Kadam, M.Sc., Ph.D. in Zoology, working as a Vice Principal and head at Gokhale education society's Art, commerce and Science college, Jawahar, district Palghar, Maharashtra. He has 25 years teaching and 20 years research experience. Dr. Kadam has published many books, book chapters research articles monograph and research papers in National and international journals. Also presented many research papers in National and international conferences, symposium and Science meets across Asia. Dr. Kadam also participated in many training programs, Science Olympiad, workshops, short term courses, social gatherings etc. he has completed many research projects funded by university grants commission, ministry of forest and environment and University of Mumbai. He is Registered Ph.D. guide in zoology of Mumbai University and 5 students are registered for PhD under his guidance. His research area are ecology of phytoplankton, zooplankton vertical migration, Marine benthic fauna, water and sediment quality, mangrove diversity, Marine microbiology, Marine biotechnology chemical stenography, climate changes, aquaculture, coastal research and Marine conservation etc. Dr.kadam is life member of Odisha environmental society , Indian Science Congress, Bangalore society of India, Bombay natural history society and society of biological chemist etc.

Ms. Ruksar is pursuing her Ph.D. in the Department of Zoology at Chaudhary Charan Singh Haryana Agricultural University, Hisar. She was awarded an HAU merit scholarship during M.Sc. degree. She has successfully qualified for the DBT-JRF 2023. She has demonstrated exceptional skills and knowledge in the field of research. Ms. Ruksar has participated and presented her research work in four international conferences and two national conferences. She has also participated in 10 days training programme on "IMC, RAS, Biofloc, Shrimp and catfish culture" at Aquaculture Research and Training Institute, Hisar. Recently, she has published a research article named "Agri-Aqua Fusion: Sustainable Integration of Fish Farming and Agriculture". She has published two research paper as a co-author. Her areas of expertise include Aquaculture and Fisheries.

Dr. Pritesh Ramanlal Gugale (M.Sc., Ph.D., B.Ed. D.M.L.T.) is currently working as assistant professor at the Department of zoology, Rayat Shikshan Sanstha's Dada Patil Mahavidyalaya Karjat Dist. Ahmednagar. He is recognised as a research Guide in Savitribai Phule Pune University, Pune and also recognised as a PG teacher in Shivaji University, Kolhapur. He has teaching experience of 11 years at Under Graduate level and 04 years at Post Graduate level in various Colleges. He has published 09 research papers (International) in UGC Care and peer reviewed journals. He conducted the Certificate Course of Basic Pathological Techniques under the Short-Term Courses in the college. He also conducted Blood and Blood Group Check-up camps for college and school students and teaching staff too.

Dr. Sachin M. Yeole (M. Sc., Ph. D.) is working as an Associate Professor of Zoology at M S P Mandal's, Shri Shivaji College Parbhani (MS). He has 15 years of teaching and research experience. He is Recognized PG Teacher and Research Supervisor in Zoology of Swami Ramanand Teerth Marathwada University Nanded since 2017. He is SPOC of SWAYAM-NPTEL Local Chapter (IIT Madras) of his college (LC 474). He successfully completed One Minor Research Project funded by UGC WRO Pune. He has attended 51 conferences of state, national & international standard and presented his research work therein. There are three edited books to his credit. He has published 32 research papers in various reputed journals as well as there are 9 articles to his account. Novel Research Academy, Puducherry honored him by conferring National Level Best Young Faculty Award.

Bhumi Publishing

ISBN 978-93-95847-28-5

9 789395 847285