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Host Modulation Therapy with Short Term Use of Oral Omega-3 Fatty Acids as an Adjunct to Non-Surgical Periodontal Therapy - A Pilot Project

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Keywords
Host modulation therapy, scaling and root planing, omega-3 fatty acids, non- surgical periodontal therapy, probing depth, clinical attachment level

Abstract
Introduction A fairly popular beneficial therapy for the non-surgical administration of periodontal infection is host modulation therapy (HMT). A commonly administered agent, Omega-3 fatty acids, otherwise called PUFA, has shown promising results of the various modalities explored in the recent years. This objective of this study was to assess its clinical impact on periodontal markers in individuals with periodontitis. Methods: For this three-month preliminary trial, scaling and root planing were performed on 60 patients with periodontitis who were between the ages of 18 and 70. From that point onward, they were randomly assigned into two groups, each with 30 patients: the experimental group, which got omega - 3 fatty acid gel capsules for a period of 2 weeks, and the control group, which got no further therapy. The clinical parameters assessed at baseline, one month, and 90 days post-introductory treatment were periodontal charting which included probing depths and clinical attachment loss; plaque index and bleeding score. Results: Despite the fact that the clinical periodontal parameters, in both the test and control groups fundamentally improved over the long run ($P < 0.001$), there were no huge contrasts between the 2 groups when compared with each other in any of the time intervals ($P > 0.05$) except for probing depths and clinical attachment levels. ($P< 0.001$). Furthermore, the experimental group's mean score contrasts were only a somewhat higher than those of the control group. Conclusion: Within the limitations of the study, Omega-3 fatty acids as a host modulation agent might demonstrate to be an economical and practical adjunct to non-surgical periodontal therapy in the treatment of patients with periodontitis.

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1. Introduction

Clinicians and researchers have long held that a patient's plaque levels essentially affect the severity and course of their periodontal infection [1]. The response of periodontal tissue to plaque changes between individuals paying little heed to how well they keep up with their oral hygiene, and a few patients might be more vulnerable to fostering the disease than others.

Despite the fact that bacterial plaque, along with other contributing factors, assists with the initiation of periodontal disease, it is not the sole reason for periodontal breakdown. The severity, rate of progression, and adequacy of treatment were completely observed to be fundamentally affected by host reaction. The previously mentioned perception prompted the utilization of host modulation therapy, an adjuvant treatment approach that Williams [2] and Golub [3] first introduced to dentistry in 1990. This strategy is utilized related to customary SRP and medical procedure to control how the host's safe framework responds to bacterial offense. Modulatory therapy depends on dealing with the host side of the host-microbes connection utilizing pharmacological medications, which thus helps bring down the levels of proinflammatory cytokines and proteins responsible for periodontal tissue loss, including as MMPs, IL-1, IL-6, PGE, and TNF-. The substances being used will likewise mirror or lead to increment in the levels of defensive/calming arbiters (such TIMPs, IL-4, IL-10, and IL-1ra).

HMTs do not impede the body's regular protective cycles; rather, they simply modify the invulnerable reaction and enhance the results of periodontal treatment. Host modulatory substances, for example, NSAIDs, bone morphogenic proteins, omega-3 fatty acids, and probiotics, can be conveyed both locally and systemically. Modulatory medications might assist with the management of different persistent incendiary conditions like joint pain and skin issues when administered systemically.

Since the body cannot create omega fatty acids, which are polyunsaturated fatty acids, they should be eaten with food [4]. Omega fatty acids arrive in different structures, including omega-3, omega-6, and omega-9. Omega-3 fatty acids, for example, alpha-linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), are available in fish oils, salmon oils, and flaxseed oils.[5] Omega-3 fatty acids (DHA, EPA) help dental health since they significantly affect oral microrganisms such as prophyromonas gingivalis, prevotella intermedia, aggregatibacter actinomycetem comitans, streptococcus mutans, and candida albicans[6][7]. Omega-3 fatty acids are connected to the phospholipids of cell layers and go about as predecessors for lipid mediators to oversee cell hailing, quality enunciation, and provocative cycles [8]. Resolvins and protections are byproducts of omega-3 fatty acids metabolism; which have anti-inflammatory and immunoregulatory effects that interferes with the production of pro-inflammatory cytokines [9][10]. Furthermore, it develops host modulation, helps increase anti-inflammtory mediators, represses osteoclast movement, and advances phagocyte action. It likewise impacts the production of PGE2, LTB4, COX-2, TNF-ALPHA, AND IL-1BETA through lipid mediators like resolvin and protectin, which are derived from the provocative mediators, eicosanoids[11]. These protectins and resolvins direct and dispose of inflammation. According to Serhant al. (2008), omega-3 works with Protectins and resolvins that help monocyte enlistment while diminishing neutrophil invasion. [12] Additionally, studies have shown that involving Omega-3 as an adjuvant to SRP and for host modulation impacts and brings down the creation of pro-inflammatory substances, thus decreasing periodontal disease aggravation.

Hence, the objective of the present study was to assess how oral omega-3 fatty acid supplementation in patients with periodontal disease impacted the clinical changes in periodontal markers.

2. Materials and Methods:

Materials

Following the approval of the Institutional research review board (IRRB-08-04082022), 60 patients (22 males and 38 females) aged between 18years to 72 years were recruited for the study conducted at the dental clinics, Ibn-Sina National College for medical sciences, Jeddah; KSA.

Patients with history of any kind of allergies, systemic diseases, pregnancy, breast feeding, smoking habits, parafunctional habits, brushing habits and any regular medication intake that may interfere with the treatment provided were excluded from the study. The

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participants included in the study consisted of patients from whom written consent was obtained and who were diagnosed with periodontal disease following clinical periodontal examination which included plaque and bleeding indices with periodontal charting.

All 60 patients underwent scaling and root planing. Proper oral hygiene instructions and re-enforcement were ensured. The patients were then randomly divided into 2 groups – test and control comprising of 30 patients each. Omega-3(180mg EPA/ 120mg DHA) premium fish oil softgels (California Gold nutrition, Madre labs, LLC, 301N. Lake Ave.#500 Pasadena, CA 91101) were administered twice a day for 14 days for the test group patients in the first visit after scaling. The control group patients were not given any medication.

A recall visit was scheduled for both the groups at the end of 1 month and after 3 months when the periodontal examination was repeated which included plaque index, bleeding index and periodontal charting.

All the data comprising of baseline , 1 month and 3 months parameters were entered into a Microsoft excel sheet and later analyzed.

Statistical analysis was performed using SPSSV22 software. Parametric tests were used to compare means within both test and control groups and also to compare the means between the 2 groups at various time intervals; baseline, 1 month and 3 months. Periodontal disease status was also assessed and reported as percentage values. Descriptive and inferential analyses were performed. Continuous variables are presented as mean±standard deviation, while categorical measurements are presented as number (percentage). Significance was assessed at a 5% level.

3. Results:

The segment data for the examination populace, which comprised of 38 females and 22 males with ages ranging from 18 to 72 years, is displayed in Table 1.

Table 1 – Demographic Data**:**

		Number	**Percentage**
GENDER	**MALES**	**22**	**36.67%**
	FEMALES	**38**	**63.33%**
AGE GROUPS	18-25	11	**18.33%**
	26-35	23	**38.33%**
	36-45	17	**28.33%**
	46-55	6	**10%**
	56-65	2	**3.33%**
	>65	1	**1.67%**

Table 2: Test Group

2A: Plaque Index:

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE- 1 MONTH	27.19667	15.40626	2.81278	21.44388	32.94946	9.669	29	.000
BASELINE –3 MONTHS	42.28000	19.46928	3.55459	35.01005	49.54995	11.894	29	.000
1 MONTH- 3 MONTHS	15.08333	7.82190	1.42808	12.16259	18.00408	10.562	29	.000

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2B: Bleeding Index:

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	12.22900	15.16969	2.76959	6.56455	17.89345	4.415	29	.000
BASELINE-3 MONTHS	17.97833	21.32450	3.89330	10.01564	25.94103	4.618	29	.000
1 MONTH-3 MONTHS	5.74933	9.26206	1.69101	2.29082	9.20785	3.400	29	.002

2C: Probing Depth:

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	.67500	.88082	.08041	.51579	.83421	8.395	119	.000
BASELINE-3 MONTHS	.80000	.88498	.08079	.64003	.95997	9.903	119	.000
1 MONTH-3 MONTHS	.12500	.33211	.03032	.06497	.18503	4.123	119	.000

2D: Clinical Attachment Loss:

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	.65000	1.00126	.09140	.46901	.83099	7.111	119	.000
BASELINE-3 MONTHS	.84167	1.15224	.10518	.63339	1.04994	8.002	119	.000
1 MONTH-3 MONTHS	.19167	.50702	.04628	.10002	.28331	4.141	119	.000

Within the test group (table 2- A,B,C&D), there was a significant reduction in the plaque index, bleeding index, probing depths and clinical attachment levels during the various time intervals from baseline to 1 month($P<0.001$), baseline to 3 months ($P<0.001$) and 1 to 3 months ($P< 0.001$)

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Table 3: Control Group**:**

3A – Plaque Index

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	42.55300	26.82311	4.89721	32.53709	52.56891	8.689	29	.000
BASELINE-3 MONTHS	36.51000	15.55622	2.84016	30.70121	42.31879	12.855	29	.000
1 MONTH-3 MONTHS	-6.04300	24.57565	4.48688	-15.21970	3.13370	-1.347	29	.188

3B – Bleeding Index

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	11.48500	16.09290	2.93815	5.47581	17.49419	3.909	29	.001
BASELINE-3 MONTHS	15.75667	20.02591	3.65621	8.27887	23.23446	4.310	29	.000
1 MONTH-3 MONTHS	4.27167	5.75373	1.05048	2.12319	6.42015	4.066	29	.000

3C – Probing Depth

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	.30000	.65594	.05988	.18143	.41857	5.010	119	.000
BASELINE-3 MONTHS	.62500	.76765	.07008	.48624	.76376	8.919	119	.000
1 MONTH-3 MONTHS	.32500	.47034	.04294	.23998	.41002	7.569	119	.000

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3D – Clinical Attachment Loss

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	.37500	.77852	.07107	.23428	.51572	5.277	119	.000
BASELINE-3 MONTHS	.70833	.98216	.08966	.53080	.88587	7.900	119	.000
1 MONTH-3 MONTHS	.33333	.53974	.04927	.23577	.43089	6.765	119	.000

However, within the control group, there was significant reduction in the plaque scores (table 3 A) from baseline to 1 month(P<0.001) and baseline to 3 months(P<0.001); but not from 1 month to 3 months(P>0.05). But bleeding scores (table 3B), probing depth(table 3 C) and clinical attachment loss(table 3D) showed significant reduction in the various time intervals(p<0.001).

Table 4 – Test V/S Control Group

4A - Plaque Index:

Matched Examples Test				
	Mean	**N**	**Std. Deviation**	**Std. Error Mean**
BASELINE-1 MONTH	23.7100	30	13.64067	2.49043
	27.1967	30	15.40626	2.81278
BASELINE-3 MONTHS	36.5100	30	15.55622	2.84016
	42.2800	30	19.46928	3.55459
1 MONTH-3 MONTHS	12.8000	30	10.68483	1.95077
	15.0833	30	7.82190	1.42808

Matched Examples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	-3.48667	20.00805	3.65295	-10.95779	3.98446	-.954	29	.348
BASELINE-3 MONTHS	-5.77000	22.91746	4.18414	-14.32752	2.78752	-1.379	29	.178
1 MONTH-3 MONTHS	-2.28333	11.79930	2.15425	-6.68926	2.12260	-1.060	29	.298

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4B - Bleeding Index

Matched Examples Test				
	Mean	**N**	**Std. Deviation**	**Std. Error Mean**
BASELINE-1 MONTH	11.4850	30	16.09290	2.93815
	12.2290	30	15.16969	2.76959
BASELINE-3 MONTHS	15.7567	30	20.02591	3.65621
	17.9783	30	21.32450	3.89330
1 MONTH-3 MONTHS	4.2717	30	5.75373	1.05048
	5.7493	30	9.26206	1.69101

Matched Examples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	-.74400	21.32939	3.89420	-8.70852	7.22052	-.191	29	.850
BASELINE-3 MONTHS	-2.22167	28.57673	5.21737	-12.89239	8.44906	-.426	29	.673
1 MONTH-3 MONTHS	-1.47767	11.46514	2.09324	-5.75882	2.80349	-.706	29	.486

4C - Probing Depth

Matched Examples Test				
	Mean	**N**	**Std. Deviation**	**Std. Error Mean**
BASELINE-1 MONTH	.3000	120	.65594	.05988
	.6750	120	.88082	.08041
BASELINE-3 MONTHS	.6250	120	.76765	.07008
	.8000	120	.88498	.08079
1 MONTH-3 MONTHS	.3250	120	.47034	.04294
	.1250	120	.33211	.03032

Matched Examples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	-.37500	.97930	.08940	-.55202	-.19798	-4.195	119	.000
BASELINE-3 MONTHS	-.17500	.94968	.08669	-.34666	-.00334	-2.019	119	.046
1 MONTH-3 MONTHS	.20000	.55911	.05104	.09894	.30106	3.919	119	.000

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4D - Clinical Attachment Level

Matched Examples Test				
	Mean	**N**	**Std. Deviation**	**Std. Error Mean**
BASELINE-1 MONTH	.3750	120	.77852	.07107
	.6500	120	1.00126	.09140
BASELINE-3 MONTHS	.7083	120	.98216	.08966
	.8417	120	1.15224	.10518
1 MONTH-3 MONTHS	.3333	120	.53974	.04927
	.1917	120	.50702	.04628

Matched Examples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
BASELINE-1 MONTH	-.27500	1.25666	.11472	-.50215	-.04785	-2.397	119	.018
BASELINE-3 MONTHS	-.13333	1.52808	.13949	-.40954	.14288	-.956	119	.341
1 MONTH-3 MONTHS	.14167	.72525	.06621	.01057	.27276	2.140	119	.034

Comparisons between the test and control group revealed no significant differences at the various time intervals(P>0.05) with respect to plaque index and bleeding index. However, there was a significant difference between the 2 groups with regard to probing depths at the various time intervals(P<0.05) as well as in clinical attachment levels from baseline to 1 month(P<0.05) and 1 month to 3 months(P<0.05). No significant change was observed from baseline to 3 months.(P>0.05)

With regard to the disease status in the test group(TABLE 5 A), there appeared to be no change in the extent of the disease with number of patients with generalized and localized form of the disease remaining nearly constant from baseline to 1 to 3 months. No change was observed in the number of patients with stage 1, 3 & 4 disease, but marginal changes in stage 2 at 3 months(50%) was observed. Additionally, slight change was observed in the grade of the disease with grade A at 3 months(6.67%) with no change observed in the grade B and C at the various time intervals. The stability status of the disease improved drastically from baseline(23.33%) to 1 month(56.67%) to 3 months(70%) whereas the unstable status also showed drastic reduction from baseline(50%) to 1 month (16.67%) to 3 months(10%). No much change was observed with the status of remission.

Nearly similar patterns were also observed in the control group(TABLE 5 B) in the extent of the disease with slight changes in the localized and generalized forms from baseline to 1 month to 3 months. Additionally, minor changes were also observed in the disease stages and grades accross the various timelines. However, significant changes were observed in the status of the disease with proportion of stable patients increasing from 30% at baseline to 63.33% at 1 month to 70% at 3 months. So also the proportion of unstable patients reduced from 30% at baseline to 13.33% at 1 month to 10% at 3 months. The proportion of patients in remission also improved from baseline (40%) to 1 month(23.33%) to 3 months(16.67%). 1 patient (3.33%) returned to the state of periodontal health.

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Table 5: Changes in Diagnosis and Disease Status

5A – Test Group

		DIAGNOSIS					
EXTENT		**BASELINE**	**%AGE**	**1MONTH**	**%AGE**	**3 MONTHS**	**%AGE**
	GENERALISED	27	90%	27	90%	27	90%
	LOCALISED	3	10%	3	10%	3	10%
STAGE	1	5	16.67%	5	16.67%	5	16.67%
	2	14	46.67%	14	46.67%	15	50%
	3	10	33.33%	10	33.33%	10	33.33%
	4	1	3.33%	1	3.33%	1	3.33%
GRADE	A	1	3.33%	1	3.33%	2	6.67%
	B	20	66.67%	20	66.67%	20	66.67%
	C	9	30%	9	30%	9	30%
STATUS	STABLE	07	23.33%	17	56.67%	21	70%
	UNSTABLE	15	50%	5	16.67%	3	10%
	REMISSION	8	26.67%	8	26.67%	7	23.33%

5B – Control Group

		DIAGNOSIS					
EXTENT		**BASELINE**	**%AGE**	**1MONTH**	**%AGE**	**3 MONTHS**	**%AGE**
	GENERALISED	24	80%	21	70%	22	73.33%
	LOCALISED	6	20%	9	30%	7	23.33%
STAGE	1	11	36.67%	11	36.67%	11	36.67%
	2	13	43.33%	15	50%	14	46.67%
	3	6	20%	4	13.33%	4	13.33%
GRADE	A	3	10%	3	10%	3	10%
	B	23	76.67%	24	80%	23	76.67%
	C	4	13.33%	3	10%	3	10%
STATUS	STABLE	9	30%	19	63.33%	21	70%
	UNSTABLE	9	30%	4	13.33%	3	10%
	REMISSION	12	40%	7	23.33%	5	16.67%

Periodontal Health – 1(3.33%)

4. Discussion:

While the presence of bacterial biofilm by all accounts, seems to be fundamental for the initiation of periodontitis, immunological host reaction is believed to be a significant component in the progression of the condition [13]. Host modulation therapy is thus presently seen as a reasonable adjunctive system. It involves adding local and systemic drugs to periodontal therapy as an adjuvant [14]. By balancing inflammation cycles, it seeks to limit tissue destruction [15]. Hypothetically, a great many drugs, including antibiotic medication, bisphosphonates, and non-steroidal mitigating meds (NSAIDs) seem suitable for use in host-modulation therapy [16][17]. However, they have undesirable impacts and must be used to a limited extent [18]. Polyunsaturated omega-3 fatty acids (PUFA) are a promising compound with regards to host modulation in numerous persistent inflammatory problems and show less undesirable

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effects. In a review of literature in 2016 [19], Chee et al. portrayed their gainful impacts on periodontal inflammation in this situation.

Clinical examinations have shown that dietary fatty acids, particularly omega-3 polyunsaturated fatty acids (PUFA), lessen disease progression in individuals. As per prevalent thinking, these significant fatty acids and their precursor, the ∝- linoleic acid tracked down in vegetable oils, work by forestalling the change of arachidonate to favorable inflammatory eicosanoids or by going about as a substitute substrate that produces less potent products. Resolvins are new oxygenated products of omega-3 PUFAs that have gone through enzymatic cycles like those that produce leukotoxin(LX). They make strong mitigating impacts. Phase - 1 therapy, the best quality level of periodontal therapy for a wide range of periodontitis, was directed to all 60 patients in our study. The purpose of our study was to look at the clinical periodontal results of one portion of the study populace, who got omega-3 fatty acid agents as an adjunct, to the next half, who got no agent administered and was simply checked for changes in periodontal parameters throughout the study period. Every single clinical parameter, including plaque index, bleeding record, probing depths, and clinical attachment levels, showed a significant improvement from baseline to 1 month to 3 months.

In various systemic conditions, like rheumatoid joint pain, cardiovascular illness, and inflammatory gut disease, as well as in a large number of animal models of inflammatory disease, the helpful impacts of dietary ω- 3 PUFA supplementation have been shown [20, 21]. However, there is a scarcity of data in regard to periodontal inflammation. As per one review, periodontitis and hyperlipidemia might be connected [22]. The enzymatic pathways of aggravation act by impeding the production of cytokines suggesting an anti-inflammatory action of ω- 3 PUFAs [23, 24].Our discoveries are upheld by various clinical investigations that show ω- 3 PUFAs may enormously reduce the inflammatory burden on the host.

It is satisfactory to utilize ω- 3 PUFAs to treat periodontal problems since they are all around endured by patients and have anti-inflammatory characteristics as shown in a study where ω- 3 PUFAs were administered to patients with ongoing periodontitis, where PD and CAL were emphatically reduced when contrasted with the placebo group [25]. Elkhouli [26] has likewise upheld these discoveries. Moreover, combined therapy with omega-3 and omega-6 PUFAs essentially diminished PD contrasted with patients who got a placebo in a clinical trial incorporating patients with persistent periodontitis [27].

While a few examinations have taken a gander at the anti-inflammatory properties of omega-3 fatty acids, there is little information accessible on their clinical effect. Omega -3 fatty acid supplementation lessens gingival inflammation in human models of gingival disease, as per Campan et al. (1996). Fish oil has also been shown by Campan et al. (1997) to decisively bring down gingival index, but they saw no variations between the test and control groups thereby concluding that Omega-3 fatty acids can reduce inflammation. These discoveries are in accordance with those of the ongoing study, affirming the positive effect of taking omega-3 fatty acids. Arachidonic acid synthesis is hindered by omega-3 polyunsaturated fatty acids, which contend with it as substrates for the cyclooxygenase and lipoxygenase pathways. Both experimental gingivitis and clinical periodontitis have responded well to medication that repress the cyclooxygenase and lipoxygenase pathways, which bring down the creation of proinflammatory arachidonic acid mediators. The modified end products of cyclooxygenase and lipoxygenase pathways, which are less provocative, are additionally delivered by the digestion of omega-3 polyunsaturated fatty acids. On the other hand, Eberhard et al. (2006), found that washing gingivitis sites with omega-6 fatty acids altogether diminished GCF when contrasted with omega-3 fatty acid. The conflict might be related to local delivery of the agents in the study.

Four of the six studies included in a systematic review by Kruse et al. (2020) showed a significant improvement in periodontal clinical parameters (PD and CAL) when periodontal therapy was done with adjuvant omega-3 fatty acids rather than a placebo. The thought of embracing host response alteration strategies in the treatment of periodontitis to stop moderate tissue weakening is upheld by predominantly ideal impact of omega-3 supplementation in PD and CAL, where an impressive decrease happened in most of occurrences, a novel impact might be shown. The meta-analysis upheld these findings within the imperatives of the information that were accessible.

The mean distinctions of the periodontal parameters were marginally higher in the experimental group contrasted with the control group, despite the fact that comparative changes were likewise found in the control group and genuinely there were no tremendous

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contrasts between the 2 groups of the study population. Both groups experienced comparative changes in disease status, with a level of stable cases ascending over the time intervals of the study and the level of unstable patients falling. However, the changes were more evident in the test group.

Omega-3 fatty acids have anti-inflammatory properties, but only one review tracked down a significant further decrease in BOP, suggesting other systemic modes of activity. Three studies uncovered a critical improvement in other periodontal measurements, supporting host-intervened dysbiosis regardless of the Plaque score. Woelber et al likewise concluded that without an adjustment of oral hygiene measures, a decrease in gingival and periodontal inflammation must be accomplished by a suitable diet which is appropriate for oral wellbeing. Since the presence of pathogenic biofilm alone does not qualify for the tissue destruction in the periodontium, the treatment of periodontal disease ought to progressively focus on host-modulation. Due to an "ongoing need" of inflammation resolution, periodontal therapies that essentially target plaque decrease need to be enhanced with adjuvant methods [28, 29, and 30].

Small sample size, lack of long term follow up and dosage alteration based on body mass index were a few of the shortcomings of this pilot project. The assessment of inflammatory markers could also have been included, which would have added authenticity to the clinical discoveries.

5. Conclusion:

In conclusion, among the many methods available to treat periodontitis, host modulation with omega-3 fatty acids dietary supplements as an adjunct to conventional nonsurgical therapy (SRP) may be important in controlling the potential deteriorative effects of the host response in patients with chronic periodontitis. This therapeutic modality may be a cheaper and safer adjunctive therapy option than currently available ones for the prevention and treatment of periodontitis.

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