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Research Article

EVALUATION OF TOTAL ORGANIC CARBON REMOVAL EFFICIENCY IN SANLIURFA DRINKING WATER TREATMENT PLANT

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Abstract: *5.8 billion cubic meters of water is drawn from the water resources in Turkey for drinking and potable water network, according to the Turkey Statistical Institute's 2016 Municipal Water Statistics results. 92.9% of this water is treated by conventional treatment methods, 6.1% by advanced treatment techniques and 1% by physical treatment methods. Conventional drinking water treatment plants generally include oxidation, pretreatment, chemical treatment (coagulation-flocculation-sludge), filtration and disinfection units. Similar treatment units are used in Sanliurfa drinking water treatment plant which is the subject of the present study. Total Organic Carbon (TOC) is one of the water quality determination methods such as BOD and COD. It refers to organic substances dissolved or suspended in water. With this study; the treatment efficiency of Sanliurfa drinking water treatment plant was evaluated according to the TOC parameter. The TOC removal efficiency performance evaluation of treatment plant was made by comparing experimental data for January-May period. According to this; average efficiency was determined as 28.51%. As a result, this performance value is concluded to be an average treatment efficiency for such conventional drinking water treatment plants.*

Keywords*: Total organic carbon (TOC), treatment system, drinking water treatment plant, Sanliurfa.*

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1. Introduction

Water is vital for human life. Most of the human body consists of water and it is impossible for human life to continue without reaching the water. Although water is at the forefront of almost all needs in human life, 1.1 billion people do not have access to healthy drinking water resources. Therefore, in order to increase the potable water resources, it is necessary to purify and improve the water from all pollutants [1]. Water resources in nature are generally not suitable for direct use as drinking water. It should, therefore, be ensured that the water is subjected to a purification process.

Surface water is the main source of drinking water in many regions of the world. High concentrations of organic carbon are a cause of concern all over the world. Because dissolved and particulate organic carbon pollutants in water are transportable and can adversely affect drinking water treatment processes [2].

A typical drinking water treatment plant generally includes oxidation, pretreatment, coagulation, flocculation, precipitation, filtration and disinfection units. Chlorine agents are used in raw water to significantly reduce the charge of pollutants in the pre-chlorination (oxidation) process [3, 4]. However, the reaction of chlorine with NOM or micro-contaminants during this process leads to the formation of carcinogenic trihalomethanes (THMs) and haloacetic acids (HAAs) [5-8]. In the coagulation, flocculation and precipitation units, the turbidity of the water is removed and then the final residues are removed by filtration. Finally, the pH of the effluent is neutralized, and a disinfection process is applied to stabilize the drinking water quality [9, 10].

In this study water samples were taken from Sanliurfa Drinking Water Treatment Plant. The total treatment capacity of the plant is 540 000 m^3/day and the hydraulic capacity is 600 000 m^3/day. As in Sanliurfa drinking water treatment plant, a conventional drinking water treatment plant generally has oxidation, coagulation, flocculation, filtration, and disinfection units.

TOC is a collective parameter used to determine the amount of organic matter in the water. It measures the concentration of organic matter in the water. Drinking water may contain organic substances below certain limits. The limit for the TOC parameter is 4 mg/L. In order to minimize disinfection by-product formation in the world, the maximum TOC concentration that should be observed at the exit of the drinking water treatment plant is limited to 4 mg per liter. According to the results of monitoring performed within the context of each of the at least one drinking water treatment plant in Turkey it has been reported to provide this value [2].

In a study carried out in terms of quality control parameters of the drinking water system of the central province of Sanliurfa, serious problems were found in the source, storage and network system [11]. In the study conducted by Kırıkçı (2006), it was aimed to observe the formation of trihalomethanes, which are known to occur after pre- chlorination and which have serious limitations in the world as a carcinogenic substance, in the water supplied from Sanliurfa Drinking Water Treatment Plant and are far below the predicted [12].

The measurement of organic carbon in natural water resources is a quick and simple method for monitoring pollution. In addition, TOC measurements of drinking water treatment plants allow the observation of the relationship between THM and organic matter in the effluent. The removal of pollutants varies according to the coagulants used in conventional drinking water treatment plants. Polialuminum Chloride Hydroxide Sulphate (PACS) is used as coagulant and polyelectrolytes are used as an auxiliary coagulant in the treatment plant.

In this study, the efficiency of the treatment plant will be determined by the analyzes to be performed at the entrance and exit of these coagulants used in the treatment plant. In addition, the

deterioration of water quality in drinking water, the formation of carcinogenic disinfection by-products by the final chlorination at the output of the treatment, and thus the removal of TOC, which causes some operational problems in the treatment system is targeted.

2. Materials and Methods

TOC parameter was measured in Sanliurfa drinking water treatment plant.

2.1. Sampling and analysis

Samples were taken from 3 different points of the plant for analysis. These points were; the inlet unit of the treatment plant (raw water), the filtrate, and the effluent of the plant (clean water). A total of 8 samples were collected at different times in January-May. The pH analyzes were carried out in situ in the plant influent and effluent of water. In this way, the changes between the parameter of TOC and raw water, filtrate, and effluent were observed, and the efficiency was calculated. The samples were analyzed with TOC device and glass sample vessels which were calibrated without any chemical treatment. The sample vessels were named Raw Water (H), Filtrate (F), and Treatment Plant Waste (Clean Water) (T). Samples were taken to the laboratory and measurements were made.

2.2. TOC analysis

TOC analysis was made as to the TOC parameter value by making necessary adjustments on the device. In the TOC method, organic substances in water are catalytically burned at high temperature (680 °C) to form carbon dioxide (CO_2). The amount of TOC in the sample was determined by measuring the CO_2. The TOC analysis was carried out within the scope of TS 8195 EN 1484 Effective Water Quality TOC Determination Method with TOC analyzer in the treatment plant laboratory. Fig. 1 shows the TOC analyzer used in the study.

Figure 1. TOC analyzer used in the study.

As a result of these analyses, the treatment efficiency and removal percentage of the TOC parameter was calculated with the formula Eq. (1) in accordance with the regulation of Quality and Treatment of Drinking Water Supply Water as follows.

$$Treatment\ Efficiency(\%) = \frac{(Influent\ concentration - Effluent\ Concentration)}{Influent\ concentration}\ x\ 100 \quad (1)$$

The limit value determined for the effluent of the TOC drinking water treatment plant is 4 mg/L and the regulation is obliged to provide this value.

3. Result and Discussion

In recent years, one of the most important problems in drinking water treatment plants is the disinfection by-products indicated by many studies such as halo acetic acid (HAA) and THM, which are formed by chlorination process by organic substances in the water and the TOC parameter. Elimination or reduction of pollution and damages is a major problem. The removal of organic matter by coagulation depends on the amount, nature and structure of the organic matter in the water environment, the coagulant to be used, the dose of the coagulant and the pH of the water [8, 14]. In many scientific studies, it was determined that TOC removal decreased with increasing alkalinity and pH value, and removal efficiency increased with increasing TOC content and amount [15].

When the results of the analyses were examined; the highest TOC value in the raw water was 4.63 mg/L in January and 3.73 mg/L on April 19. The TOC value obtained in the filtrate was 3.41 mg/L in January and 2.73 mg/L in March. The arithmetic mean of all analyses for the filtrate was found to be 3.09 mg/L. The highest value of TOC in the effluent was 3.36 mg/L in January and the lowest value was 2.37 mg/L on February 7. The average TOC value of raw water was measured as 4.11 mg/L. The average of TOC in the effluent was 2.94 mg/L.

When the raw water in the treatment plant inlet unit and the pH values at the effluent of the plant were examined, it was observed that there was no significant decrease and increase. It was evaluated that there was a decrease in four of the measurements taken and an increase in four of them, and the mean values of raw and treated water values of this parameter were 7.47 and 7.55. The pH of the raw water was measured as 7.11 in May and 7.83 in January. It is seen in Tab.1 that the maximum pH of the treated effluent is measured in 8.16 in February and 7.15 in late January.

Table 1. Treatment Plant pH and TOC analyses results.

Measurement Number	Date	pH raw water	pH effluent	TOC raw water (mg/L)	TOC filtered (mg/L)	TOC effluent (mg/L)
1	19.01.2018	7,83	8,1	4,63	3,41	3,36
2	31.01.2018	7,56	7,15	4,18	2,85	2,91
3	07.02.2018	7,67	8,16	4,02	2,75	2,37
4	28.02.2018	7,39	7,27	4,12	3,15	2,85
5	15.03.2018	7,32	7,26	3,81	2,73	2,65
6	03.04.2018	7,46	7,33	4,23	3,25	3,18
7	19.04.2018	7,44	7,61	3,73	2,86	2,9
8	08.05.2018	7,11	7,52	4,12	3,71	3,26
Average Values		7,47	7,55	4,11	3,09	2,94

The change of TOC values in raw water, filtrate and effluent are given in Fig. 2. It is seen that the value of raw water and treated water (at the filtrate and effluent) changes according to the direction of treatment flow decreasing. In addition, the filtrate and final effluent concentration values of TOC were found to be parallel and close to each other.

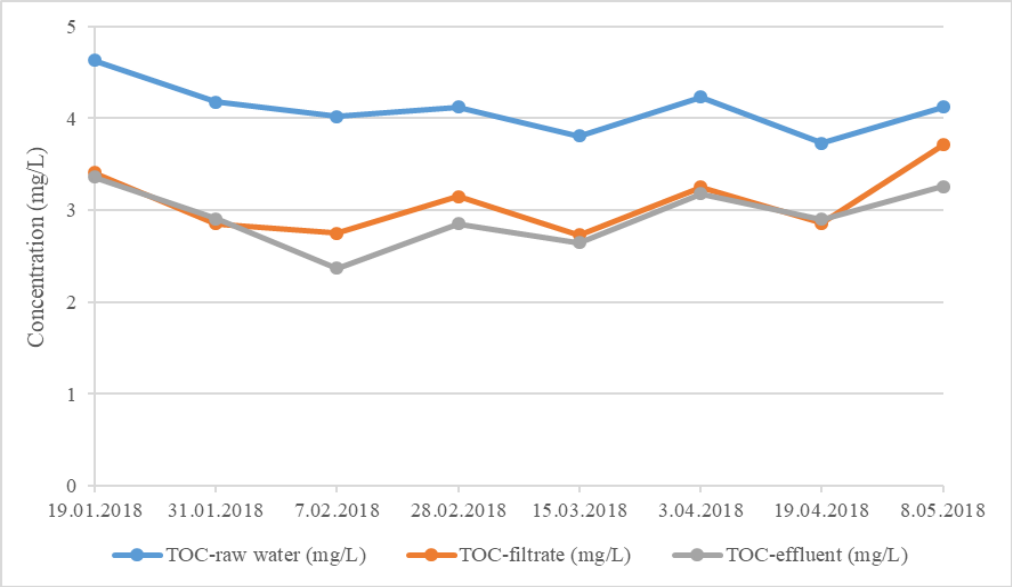

Figure 2. Variation of TOC analyses results with sampling times.

3.1. Calculation of Treatment Efficiency

The treatment efficiency of the Sanliurfa treatment plant, the results of TOC analysis the influent, the filtrate, and the effluent are given in Tab. 2 below.

Table 2. TOC analyses results and treatment efficiencies in filtrate and effluent.

Measurement Number	Date	TOC raw water (mg/L)	TOC filtered (mg/L)	TOC effluent (mg/L)	Efficiency filtered (%)	Efficiency effluent (%)
1	19.01.2018	4,63	3,41	3,36	26,35	27,43
2	31.01.2018	4,18	2,85	2,91	31,82	30,38
3	7.02.2018	4,02	2,75	2,37	31,59	41,04
4	28.02.2018	4,12	3,15	2,85	23,54	30,83
5	15.03.2018	3,81	2,73	2,65	28,35	30,45
6	3.04.2018	4,23	3,25	3,18	23,17	24,82
7	19.04.2018	3,73	2,86	2,9	23,32	22,25
8	8.05.2018	4,12	3,71	3,26	9,95	20,87
Average Values		4,11	3,09	2,94	24,76	28,51

When the treatment efficiency between raw water and filtrate and effluent summarized in Tab.2 was examined, the lowest amount of TOC was measured in May (9.95%) and the highest in January (31.82%). It can be seen that TOC treatment efficiency at the effluent is measured as 20.87% in May and highest in February (41.04%). The lowest treatment efficiency in May was 9.95%.

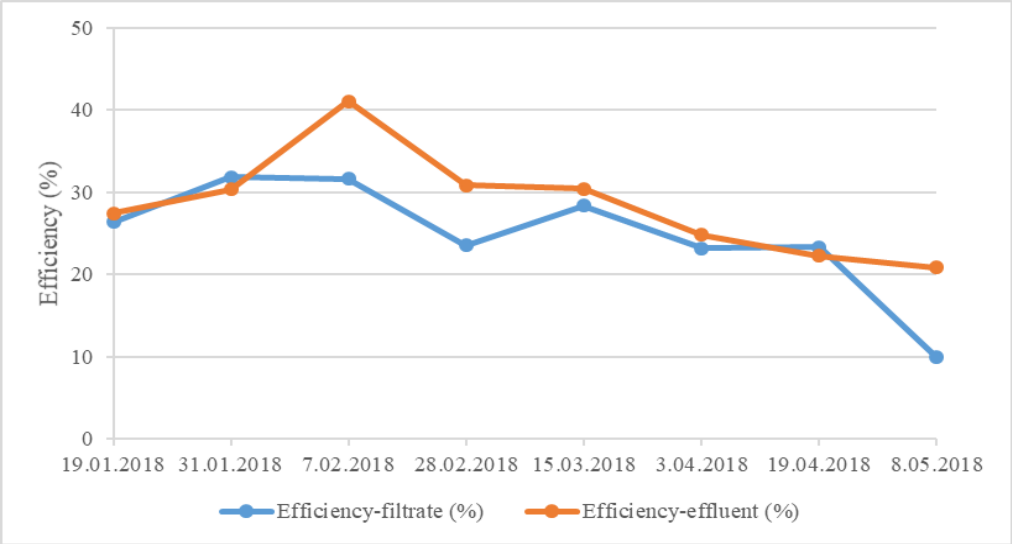

Figure 3. Variation of TOC efficiency percentage of treatment over time.

It is seen in Fig. 3 that the percentage of efficiency of the treatment in terms of TOC decreases over time, especially towards February-May. It has been evaluated that the reduction of treatment efficiency can be explained by the increase in water temperature.

4. Conclusion

Treatment efficiency of drinking water treatment plant in terms of TOC showed that; the average of all measurements at the filtrate was 24.76% and the TOC treatment efficiency of the effluent was 28.51%. The mean value of the TOC parameter was 2.94 mg/L at the effluent and it was found to provide the standard of 4 mg/L. However, it has been determined that TOC measurements and attempting to be below the standard value should be done in order to stay on the safe side for the control of disinfection by-products. It has been evaluated that this situation can be achieved by using coagulants in optimum treatment according to TOC value in raw water considering the TOC efficiency of treatment.

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Research Article

EFFECT OF DIETARY SUPPLEMENTATION OF DIFFERENT MULTI-ENZYMES ON PRODUCTION PERFORMANCE AND EGG QUALITY CHARACTERISTICS IN LAYING HENS

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Abstract: *The purpose of this study was to determine the effects of dietary addition of multi-enzymes in a different source of grains based diets on production performance and egg quality characteristics of laying hens. A total of 225, 24 weeks of old laying hens (Atak-S) were divided into 3 treatments with 5 replicates and 15 hens per replicate for 9 weeks. The control group was fed basal diet (without any supplementation) and treatment groups were fed basal diet supplemented 0.1% Enzyme-A (xylanase, ß-glucanase, cellulase, α-amilase, and protease) and 0.05 % Enzyme-B (xylanase, ß-glucanase). Productivity performance and egg quality parameters were checked weekly throughout the experiment. Dietary multi-enzymes supplementation significantly changed the shape index, yolk index and yolk color (L and a) at different weeks of trial (P<0.05). However, daily feed intake, egg production, average of egg weight and feed conversion rate were not affected by the addition of both multi-enzymes throughout the experiment. (P>0.05). Also, dietary addition two types of multi-enzymes did not affect the egg specific gravity, eggshell thickness, eggshell rate, albumen index, haugh units (P>0.05). As a result, the dietary addition of multi-enzyme did not affect the performance parameters but caused limited changes on some egg quality characteristics. of laying hens between 24-33 weeks of age.*

Keywords: *egg quality, laying hens, multi-enzymes, performance*

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1. Introduction

Animal products play an important role in providing adequate and balanced nutrition that people need and maintaining the healthy lives of individuals. Therefore, one of the most consumed animal products is the egg. Eggs are the only food source that contains all the nutrients that human needs after breast milk. Especially the rich content of essential amino acids makes the egg an accepted source of quality animal protein [1]. As a matter of fact, the biological availability of egg protein is 100%; while this value is 85% in milk, 76% in fish and 74% in beef. With increasing interest in healthy and balanced nutrition in recent years, daily egg consumption per person has also increased. Therefore, with the increase in egg consumption and consumer awareness, the concept of egg quality has started to gain importance.

In determining the quality characteristics of the egg, many criteria related to external and internal quality are taken into consideration. Egg quality which is extremely important for the consumer, as well

as the producer, is determined by; egg weight, shell thickness, egg shape, shell pore and shell color [2]. In addition to this, the shape index, specific gravity, fracture strength, surface area, and fracture-crack egg ratio also play a decisive role in external quality. Although egg weight and size vary depending on genotype, it is also influenced by feeding and environmental factors [3]. Poor eggshell quality is an important problem that negatively affects egg production, reduces hatchability and increases embryo mortality. On the other hand, egg quality with low shell strength and high risk of infiltration of pathogenic bacteria is not preferred by consumers. Therefore, egg production with high shell defects breaks consumer perception of trust and leads to economic losses. In the studies, it has been reported that the use of feed supplementation added to the mixed feed is beneficial in improving egg quality and gives positive results [4]. After commonly used enzyme studies, such as xylanase, amylase, cellulase, and phytase, which affect a single substrate in poultry nutrition, it has been reported that the use of multienzyme acting on more than one substrate may further improve feed conversion rate and performance [5]. In a study, it was reported that the addition of a multienzyme has positive effects on the rate of feed conversion in laying hens, ether extract digestibility of non-starch polysaccharides (NSP), eggshell quality and n-3 fatty acid accumulation in the egg [6]. Lima et al. [7] reported that the addition of enzyme complex to mixed feed improves intestinal health and performance in laying hens and improves internal and external egg quality. The aim of this study is to determine the effects of adding different multienzyme to laying hens mixed feed on performance (feed intake, feed conversion rate, egg production, average egg weight), egg external quality (shape index, shell rate, shell thickness, specific gravity) and egg internal quality (yolk index, albumen index, Haugh unit and yolk color and blood and flesh stain) characteristics.

2. Materials and Methods

A total of 225, 24 weeks of old laying hens (Atak-S) were divided into 3 treatments with 5 replicates and 15 hens per replicate for 9 weeks. The control group was fed basal diet (without any supplementation) and treatment groups were fed basal diet supplemented 0.1% Enzyme-A (xylanase, ß-glucanase, cellulase, α-amilase, and protease) and 0.05 % Enzyme-B (xylanase, ß-glucanase). Performance and egg quality parameters were checked weekly throughout the experiment. Each kg of an enzyme A used in the assay contains 250.000 U + 790.000 EPU of xylanase, 1.000.000 U + 8.700 EPU of Beta-Glucanase, 350.000 U + 18.000 EPU of Cellulase, 350.000 U + 21.000 EPU of Alpha-Amylase and 7.500.000 U + 8.000 EPU of Protease. Each kilogram of enzyme B contains 200 U of xylanase and 138 U of Beta-Glucanase. The feed raw materials to be used in the study were obtained from a commercial feed factory and the mixed feeds of the experiment were prepared at the feed production facility of the Department of Animal Science, Agricultural Faculty of Dicle University. Enzyme A and B feed additives were added to the main mixed feed after pre-mixing.

The nutrient contents of the compound feed to be used in the experiments were prepared in accordance with the nutrient requirements of laying hens reported in NRC, 1994 [8]. The composition (%) and nutrient contents of the mixed feeds used in the study are shown in Table 1. The study was carried out in the Enriched Cage System in the laying hens experimental unit in the Poultry Research and Application Facility, Department of Animal Science, Agricultural Faculty of Dicle University. The enriched cage system has 3 floors and 5 cage sections on each floor. The animals in each cage compartment were group fed, and the feed and water were presented as *ad libitum.* Illumination of the experimental room was provided by fluorescence and 8 hours of dark and 16 hours of light program was applied daily. Determination of nutrient contents of feeds used in the experiment (except crude cellulose)

was performed according to the Weende analysis method [9] and the determination of crude cellulose according to the Lepper method. In the calculation of metabolic energy content of feeds, regression equation no. 9610 proposed by TSE was used [10].

Table 1. Ingredients and chemical composition of experimental diets (as-fed basis)

Ingredients	(%)
Corn	37.0
Soybean Meal (44% CP)	9.50
Full Fat Soybean	17.0
Sunflower Meal (32% CP)	13.0
Wheat	17.0
Dicalcium Phosphate (DCP)[a]	1.85
Calcium Carbonate	8.60
NaCI	0.30
Vitamin+ Mineral Premix[b]	0.25
Chemical Analysis	***(%)***
Dry Matter	90.70
Crude Ash	10.46
Crude Protein	17.00
Ether Extract	4.10
Calculated values	
ME (kcal/kg)	2744
Calcium (%)	3.81
Available Phosphor (%)	0.40
Na (%)	0.17
L-lysine (%)	0.78
Methionine+Cysteine (%)	0.59
Treonin (%)	0.61
Tryptophane (%)	0.21
Linoleik asit (%)	2.90

[a] Premix supplied per 1 kg; Calcium 24,5%, Phosphor; 18%.

[b] Premix supplied per 1 kg: vitamin A; 12.000.000 IU; vitamin D3; 2.500.000, vitamin E; 30.000 mg, vitamin K3; 4.000 mg; vitamin B1; 3.000 mg, vitamin B2; 7.000 mg, vitamin B12; 5.000 mg, vitamin B6; 5.000 mg, vitamin C; 50.000 mg, Niacin; 30.000 mg, Cal-D-Pantothenate; 10.000 mg, Biotin; 45 mg, Folic acid; 1.000 mg, Choline Chloride; 200.000 mg, Xanthate;1.500 mg, Manganese; 80.000 mg, Iron; 60.000 mg, Zinc; 60.000 mg, Co; 5.000 mg, Iodine; 1.000 mg, Cobalt; 200 mg, Selenium;150 mg.

At the beginning of the experiment, all hens were weighed and were left in the experimental group cages as their live weight and egg yield levels same. During the experiment, the egg production, feed intake and egg weight of the animals were measured weekly and feed conversion rate was calculated using the data obtained. Feed Conversion Rate (FCR) = Feed Consumption (g)/Total Egg Weight (g). Internal and external quality analyses were performed on 15 eggs collected weekly from each group on the same day. Egg weight was determined by weighing with precision balance (0.01g) every other day. Egg Shape Index (ESI): The width and length of the egg were measured by digital caliper and calculated

using the formula ESI = (Width of egg / Length of the egg) x 100. Egg Specific Gravity was measured with a density analyzer consisting of precision balance, beaker, and apparatus. For this purpose, the weight of the eggs which were kept at room temperature for 24 hours was first weighed in the air and then the weight in the water has an average temperature of 20-22 ^{o}C was calculated to determine the specific gravity of the egg.

The shells taken from the middle parts of the broken eggshell under laboratory conditions were measured by digital micrometer after drying and separating the membranes. The shelling rate was determined by the ratio of the value of the eggshells obtained with the precision balance after the membrane was removed and dried to the egg weight. Egg yolk color was determined by digital colorimeter (Minolta CR-300) in *L**, *a** and *b**. Albumen Index (AI): For this purpose, the eggs were broken on a clean glass so that they could not spread and then the albumen breadth and albumen lengths were measured using a digital caliper. The height of white was measured with digital foot micrometer and calculated with the formula AI = [albumen height (mm) / ((albumen length (mm) + albumen width (mm)) / 2)] x100. Yolk Index (YI): The diameter of the egg yolk was measured by digital caliper and the height was measured by digital foot micrometer and it was determined by the formula; YI = [(Yolk height / Yolk diameter) x 100]. Haugh Unit was calculated by using the egg weight and albumen height and by using formula; Haugh Unit = 100 Log (H + 7.57-1.7G 0.37). H: Albumen height (mm), G: Egg weight (g). Statistical analysis of the data obtained at the end of the experiment was performed using the SPSS 18.0 package program [11]. The analysis of variance of the averages was performed with the General Linear Model (GLM) ANOVA. Tukey's multiple comparison test was used to compare the differences between means.

3. Results and Discussion

No difference was found between the groups in terms of productivity performance (average feed intake, feed conversion ratio, egg production, and egg weight) in 24-33 week periods (P> 0.05) (Table 2). The findings obtained from this study were found to be consistent with some of the previous literature and it was observed that the addition of a single enzyme or multi-enzyme did not change feed intake in laying hens [12]. However, the addition of multi enzymes [13] and, phytase [14] increased feed intake. On the contrary, Torki et al. [15] reported that the addition of β-glucanase and xylanase or β-mannose-containing enzymes reduced feed intake. In another study, it was reported that the addition of protease to protein-restricted diets did not affect treatments in terms of feed intake [15]. Since the enzymes do not have an aromatic taste and are not used for appetizing purposes, it can be said that their addition to diets is not expected to have any effect on feed intake. However, it is thought that the discrepancy between the literature is affected by factors that change the efficiency of the enzyme such as laying hens, dietary raw material types, environmental and climatic conditions used in the studies, and changes in the feed intake.

Table 2. Effects of multi-enzyme supplementation in laying hens diet on performance parameters (age of 24-33 weeks)

Parameters	Groups			SEM	P-value
	Control	Enzyme A	Enzyme B		
Feed consumption, g/day	117.7	117.1	117.5	0.182	0.310
Feed conservation ratio	2.4	2.4	2.4	0.020	0.779
Egg production, %	86.4	86.4	87.1	0.771	0.211
Egg weight, g	55.4	55.8	55.2	0.174	0.335

The differences between means in the same row with different letters are significant ($P < 0.05$). SEM: Standard Error of Mean

Some researchers [15,16,17] reported that the addition of multi-enzyme improved the feed conversion rate in laying hens. In contrast, some researchers found the opposite results [18, 19, 20]. As it is known, 'FCR' is the ratio of feed intake to egg weight in laying hens. There are similarities between these results with our results. It is seen that the literature on the effect of enzyme addition to laying hens diets on egg production is incompatible. In related studies [12, 13, 19, 20] reported that the enzyme addition did not change egg production. On the other hand, Khan et al. [21] reported that dietary enzymes increased egg production in laying hens. Differences between the results may be due to the level of difference of enzymes added to the feed. There was no difference between treatment groups with respect to the average egg weight ($P > 0.05$). These results are in agreement with the results of other researchers [12, 13, 22]. The effects of multi-enzyme supplementation in laying hen diet on external and internal egg quality characteristics are given in Table 3.

Table 3. Effects of multi-enzyme supplementation in laying hens diet on external and internal egg quality characteristics (age of 24-33 weeks)

Measurements	Groups			SEM	P-value
	Control	Enzyme A	Enzyme B		
Shell rate, %	12.0	11.8	12.1	0.090	0.299
Shell thickness, mm	0.37	0.37	0.37	0.002	0.923
Specific gravity, g/cm^3	1.08	1.07	1.08	0.003	0.085
Shape index	77.2^a	76.5^b	75.9^c	0.170	0.017
Yolk index	48.1^a	46.9^b	46.7^b	0.250	0.040
Albumen index	3.9	3.8	3.7	0.050	0.333
Haugh unit	74.6	73.4	73.4	0.520	0.569
L* value	52.4	51.0	49.6	1.068	0.177
a* value	19.4	17.3	17.1	0.643	0.275
b* value	31.2	30.1	29.4	0.504	0.342

The differences between means in the same row with different letters are significant ($P < 0.05$). SEM: Standard Error of Mean.

The results showed that the dietary addition of either enzyme A or enzyme B decreased shape index value when compared with the control group ($P<0.05$). However, the egg quality indices such as eggshell

thickness, shell rate, specific gravity, albumen index, yolk colors, and blood spots were not affected by the diets (P>0.05). Similar results have been reported by various researchers [13, 15, 21]. Contrary Yaghobfar [23] reported that the addition of ß-glucanase and xylanase to barley-based diets reduced the eggshell thickness in laying hens. These different results may be due to the content or levels of enzymes used in feed.

Conclusion

In conclusion, our results showed that the addition of different multi-enzyme sources to mixed feeds did not affect the production performance of hens aged between 24-33 weeks and no changed egg quality characteristics tested except egg shape and yolk index

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Research Article

EFFECT OF ELECTROMAGNETIC FIELD ORIGINATING FROM HIGH VOLTAGE LINES ON MALONDIALDEHYDE LEVEL

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Abstract: *Our study aimed to investigate the effect of electromagnetic fields originating from high voltage lines on serum malondialdehyde level of male rats wistar albino. A total of 32 rats were randomly assigned to study in 4 groups. Groups were; Group 1: high voltage, Group 2: high voltage+ganoderma l., Group 3: high voltage+melatonin, Group 4: control. Experimental groups were exposed to high voltage for 8 hours daily for 52 days. The electric field and the magnetic field were measured. Ganoderma was administered 20 mg/kg/day as a gavage and melatonin intraperitoneally as 10 mg/kg/day. In the study, the malondialdehyde (MDA) levels of the experimental and control groups were compared. There was no significant difference between the groups. According to the control group, it was found that the MDA level of the high voltage group increased, while the Ganoderma and Melatonin groups had a small decrease at the MDA level. These results show that electromagnetic fields originating from high voltage increase the MDA serum level, which is found to decrease in the presence of ganoderma and melatonin.*

Keywords: *Malondialdehyde, High voltage, Melatonin, Ganoderma, Electromagnetic field*

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1. Introduction

With the development of technology, the use of electric vehicles has become increasingly widespread, so pollution arising from very low-frequency electromagnetic fields is gradually increasing [1]. Electromagnetic fields related to the increase in mobile phones, televisions, computers, electrical appliances and occupational exposures used in the home and business environment and negative effects on the human body have caused worries and problems. Most of the studies have shown that these areas are a negative effect, but some studies report that these areas have a positive (therapeutic) orientation [2]. There are studies of how long-term exposures of very low-frequency electromagnetic fields can reason oxidative DNA harm [3]. The main source of malondialdehyde is lipid peroxidation of polyunsaturated fatty acids. In most studies, malondialdehyde is used because it is a natural marker for oxidative stress. Several malondialdehyde methods have been developed since 1960 to determine oxidative stress in vivo and in vitro studies [4]. The polysaccharides obtained from Ganoderma lucidum showed free radical cleansing, anti-angiogenic, anti-tumor and immunosuppressive properties [5]. Melatonin has the ability to neutralize oxidative stress-related tissue damage and free radicals [6].

2. Materials and Methods

This work was conducted at the Dicle University Health Sciences Research Center. Permission was obtained from the Ethics Committee of the University of Dicle Animal Experiments (DUHADEK: 2013/13) and the study continued according to the standards set out in the Helsinki Declaration.

2.1. Animals and experimental protocol

A total of 32 rats (weighing 320,45±6,84, 4-5 months) were randomly assigned to study in 4 groups. Groups were; Group 1: HV, Group 2: HV+GI, Group 3: HV+MEL, Group 4: Control. Experimental groups were exposed to high stress for 8 hours daily for 52 days. All animals were kept in suitable environmental conditions (constant temperature of 23±1 ºC and 45-55% humidity of the air), 12 hours of the night and 12 hours of light daylight, respectively, normal food (ad-libitum) and water were always available in the animals' cage. To create electromagnetic fields, two transformers that produced 10 kiloVolt (10,000 V) of high voltage were used. For the first transformer, the input was 220 Volt, and the output was 10 kiloVolt. For the second transformer, the input was 10 kiloVolt, and the output was 220 Volt and 5,000 Volt Amper. The electric field (80,3 V/m) and the magnetic field (2,48 μT) were measured. Ganoderma (Gano Excel, Industries Sdn. Bhd., Kedah, Malaysia) extracts were prepared with distilled water with appropriate standards. Melatonin (Merck KGaA, Germany) was prepared according to the weights of rats and then dissolved with pure ethanol and dissolved in distilled water at appropriate ratios. Ganoderma was given as 20 mg/kg/day as a gavage and melatonin were administered intraperitoneally as 10 mg/kg/day. Electromagnetic field measurement The Spectran NF5035 (AARONIA AG, Strickscheid, Germany), the instrument was taken in 6-minute measurements as determined by ICNIRP.

2.2. Blood collection and detection of MDA

After the study, rats were anesthetized by administering ketamine-xylazine at appropriate ratios, blood samples taken from the sacrificed rats were studied. Blood samples were immediately centrifuged at 500 rpm for 5 minutes to collect serum for study. Assay protocol MDA-TBA (Thiobarbituric acid) (Product NWK-MDA01, Northwest, Life Science Specialties, Vancouver); Add 10 μL BHT Reagent to microcentrifuge vial. Add 250 μL calibrator or sample to the vial. Add 250 μL Acid Reagent to the vial. Add 250 μL TBA Reagent to the vial. To make a homogenous structure. Vortex V1 Plus vigorously (5-count) (Boeco, Germany). Then, Incubate 60 minutes at 60 ºC (Nuve bath, Germany). Centrifuge at 10,000 rpm for 2-3 minutes (M-240R, Boeco, Germany). Transfer reaction mixture to the cuvette. MDA serum samples were read on a UV-VIS spectrophotometer at 515 nm. (UV-1208, Shimadzu, Japan) Then Perform 3rd derivative analysis. The results were taken as μL [7].

2.3. Statistical analysis

The Kolmogorov-Smirnov test was used to assess whether the data in our study were in the normal distribution. Our test results show normal distribution. Since the number of observation is small, we used the Kruskal-Wallis test and Mann Whitney U test to evaluate the data statistically. $p<0.05$ was considered statistically significant. Explanatory statistical evaluations such as mean, standard deviation, minimum, maximum, median and p significance were determined. For these analyses, the SPSS 21. 0 (IBM SPSS Statistics for Windows, version 21.0, Armonk, NY: IBM Corp., USA) package program was used.

3. Results

In the study, the MDA levels of the experimental and control groups were compared. There was no significant difference between the groups (>0.05). According to the control group, it was found that

the MDA level of the high voltage group increased, while the Ganoderma and Melatonin groups had a small decrease at the MDA level. MDA values of all groups are shown in Table 1. In Table 2, binary group comparisons were made and it was found that there was a significant change between group 1 and group 2 and between group 1 and group 3. The addition of melatonin and Ganoderma when compared with the high voltage group gave a statistically significant result (<0.05). In Figure 1, MDA concentration levels of all groups are given graphically together.

Table 1. MDA levels of all groups

Groups / 52 day	**Mean+ SD**	**Median**	**Min**	**Max**
Group 1 HV	12,34±1,95	11,90	9,43	15,52
Group 2 HV+GL	9,27±2,46	10,25	5,27	11,55
Group 3 HV+MEL	9,09±3,65	9,37	2,86	13,54
Group 4 Control	10,90±1,85	11,37	7,74	12,67
P	P:0,073 *Chi-square:6,981*			
	P>0,05 df: 3			

Values are given as mean ± standard deviation (SD), Median, Minimum (Min) and Maximum (Max). P> 0.05 was not found significant with kruskal-wallis anova test.

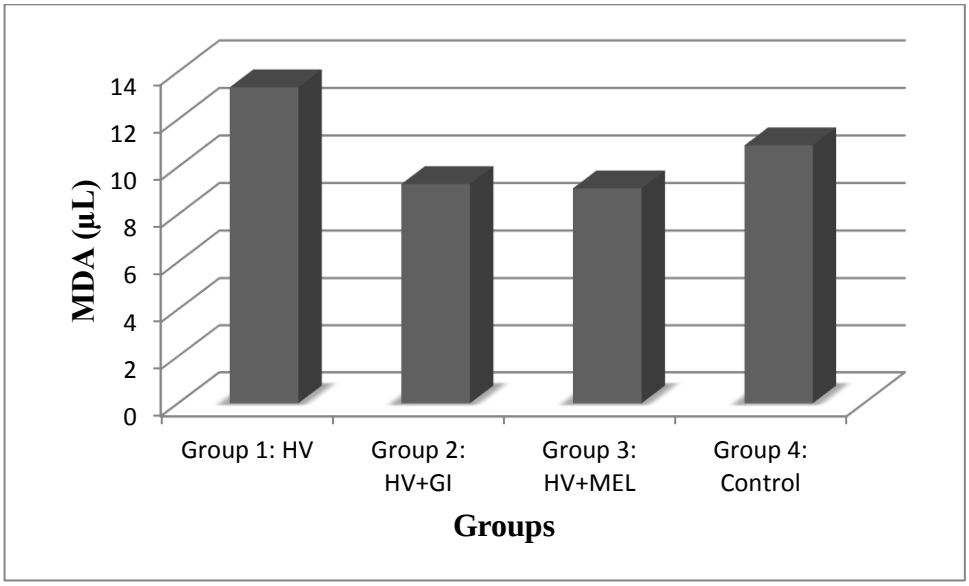

Figure 1. Graphical representation of MDA serum concentrations of rats. MEL: melatonin, HV: high voltage, GI: Ganoderma lucidum

Table 2. Binary comparisons of all groups

Groups	**P**
Group 1-Group 2	P:0,015 P<0,05*
Group 1-Group 3	P:0,046 P<0,05*
Group 1-Group 4	P:0,343 P>0,05
Group 2-Group 3	P:0,752 P>0,05
Group 2-Group 4	P:0,172 P>0,05
Group 3-Group 4	P:0,293 P>0,05

Binary group comparisons were made by Mann Whitney u test.
* P <0.05 was statistically significant in the binary comparisons.

4. Discussion

In a study conducted by Meral et al. [8], 900 MHz electromagnetic field was found to produce oxidative stress on guinea pigs brain tissue and blood parameters and it was found that the experimental group increased statistically compared to MDA level control group ($p<0.05$). In the groups exposed to 50 Hz and 80 Gauss electromagnetic fields, the MDA level of the experimental group was found to increase and the total antioxidant level decreased [9]. Magnetic field (50 Hz MF of 1.5 mT) continuous (4 h/day) and intermittent (two h/2h) exposure were found to alter antioxidant status in different tissues and significantly increase malondialdehyde level of intermittent exposures [10]. In the study conducted by Türközer and his colleagues on the Guinea pig, it was seen that the average of MDA increased with the increase of electric field compared to the sham group [11]. Çelik and colleagues investigated the effect of hairdryers device (ELF-EMA) on serum MDA used in hairdressing salons. From the serum, the oxidative effect of the ELF electromagnetic field was reported to be caused by an increase in the MDA level of the experimental group [12]. Similarly, Kula and his colleagues investigated the effect of electromagnetic fields on workers in the steel industry for 3-10 years. In the study data, it is seen that there is an increase in the plasma MDA level with the increase of the study period according to the control group [13]. In our study, it was observed that the MDA level was higher in the high voltage group than in the control group. In our study, MDA level in high voltage+melatonin and high voltage+ganoderma groups were found to be closer to the control group and decrease with respect to the high voltage group. In our study, we have determined that high-voltage electromagnetic exposures can change MDA level. Our work is consistent with the literature.

Melatonin, secreted from the pineal gland, has so far proved to be a good antioxidant. At the same time, many studies have reported that melatonin is protective against oxidative stress [14, 18, 19, 20]. Researchers investigated the effect of melatonin on lipid peroxidation during radiotherapy in female rats. Blood and tissue malondialdehyde level of radiotherapy groups were found higher than the control group. MDA levels in melatonin-treated groups showed a decrease compared to the radiotherapy group. These results report that melatonin reduces MDA levels [15]. Liver-tumorled mices were exposed to 6 Gy gamma radiation. They reported that ganoderma given to mices may have an anti-tumor effect [16]. Researchers used ganoderma extracts that have an antioxidant effect on cardiac toxicity. The study results reported that ganoderma may have cardioprotective effects. Thus, it has been found that Ganoderma lucidum inhibits lipid peroxidation and significantly reduces malondialdehyde (MDA) formation [17]. Statistical binary comparisons of our study showed a significant difference between Group 1 (high voltage) - Group 2 (voltage + ganoderma) and Group 1 (high voltage) - Group 3 (high

voltage + melatonin) ($p<0.05$). This statistical difference suggests that malondialdehyde levels may change when added with ganoderma and melatonin, and the harmful effects caused by oxidative stress can be repaired.

5. Conclusion

In our study, ELF-EMA exposure is considered to increase lipid peroxidation and oxidative stress. This effect appears to originate from the electromagnetic field originating from high voltage. Thus, after the addition of Ganoderma and Melatonin gave to the groups, the increased level of MDA was partially reduced, indicating that melatonin and ganoderma had a protective effect.

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Disclosure statement

The author states that there is no conflict of interest

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Research Article

IMPROVEMENT OF SALINE HARRAN SOILS BY ELECTROKINETIC REMEDIATION METHOD

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Abstract: *Soil salinity has become one of the environmental issues around the world. The most typical example in Turkey was the soil salinization in Harran and Akçakale Plains in recent years. Especially the Harran Plain has the big potential of water and land resources. Irrigated farming is common in the plain. Excessive and uncontrolled irrigation contributed to the high groundwater level and soil salinity in the region. The deficiency of the drainage network in the farmlands was the most important factor in the salinity. In-field drainage systems are newly established. In some areas, irrigation is conducted through the main drainage channel with high salt content (13.5 dS/m). On the other hand, pumping irrigation waters in Akçakale region have high salt content. This problem has grown especially when dealing with low-permeable soils where conventional remedial techniques are inadequate and often ineffective. Electrokinetic treatment is an innovative and clean technology for the restoration of saline soils. This process has proven to be the most effective, promising technique for the optimal and sustainable improvement of salt-affected soils.*

Keywords*: Soil salinity, Harran plain, electrokinetic remediation.*

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1. Introduction

Every year, 10 million ha of land becomes unusable due to salinity. Chlorine, sulfate, sodium, calcium, and magnesium are the main elements causing salinity, especially in arid and semi-arid climates. Especially in arid and semi-arid climatic zones, the soluble salts which are washed and mixed into the groundwater are brought to the soil surface by capillarity together with the high groundwater level and accumulation of salts occurs on the soil surface by evaporation of water [1, 2]. This accumulation takes place on the soil surface or just below the surface due to the high-temperature effect. Inaccurate irrigation practices can also lead to salinity, especially in areas with poor drainage conditions [1].

The most typical example of the soil salinization in recent years was the Harran plain. Harran plain is a very old agricultural area and important crops have been harvested in the plain from past to present. However, some of the farmlands in the area have been turned into barren lands due to salinization. For this reason, it is very important to remediate and economically evaluate the saline soils [3].

Many conventional treatment methods for soil salinization are based on irrigation, soil deposition, salt storage plants and soil changes [4-6]. Also, oxidation, ion exchange, and precipitation, photolysis, dechlorination, soil vapor extraction, soil washing, soil flushing, soil substitution mixing and exchanging saline soil with clean soil and phytoremediation methods are used in desalination. However, some of these techniques are expensive and relatively consuming methods, requiring large quantities of cleaned water and fresh soil and they cannot remove all salts from the land [7, 8]. Although the cultivation of salt accumulating plants is temporarily effective, it only reduces the intake of salt. As a result, a large amount of salt remains in the porous medium [9]. Bioremediation and chemical restoration are less effective, time-consuming and extremely expensive [10, 11]. Recently, the Electrokinetic (EK) technique has removed salts from saline soil in a laboratory scale [12-14] and pilot-scale works [15-17]. Many studies on desalination using EK remediation have been reported [18-20]. The electrochemical treatment (ECT) is an innovative, sustainable and low-cost method that has been proven for its feasibility and potential for reclaiming low permeable saline soils [21-23]. In this study, it is tried to explain the applicability of the electrokinetic remediation method in the saline Harran Plain soils.

2. Salinity in Harran Plain Soils

Harran plain is the most typical example of soil salinization in recent years (Fig. 1). The area has a semi-arid climate and poorly drained soils because of the high (>60%) clay content. Clayey soil texture has a significant risk for salinization. High clay content negatively affects the permeability of the soil and prevents the movement of water and air in the soil. Low permeability capacity increases the risk of salinization. Harran plain soils are rich in lime and potassium and are poor in nitrogen, phosphorus and organic matter. There is 24% lime in the upper soils and 26% in the lower soils. The pH of the soils varies between 7.5-8.0.

Soil salinity problems occurred due to excessive irrigation by farmers with little or no irrigation experience, topography, soil characteristics, climatic conditions, and insufficient drainage problems. Irrigated farming continues within the scope of GAP, 150,000 hectares of Harran plain lands. Clayey soil texture has a significant risk for salinization. Today, approximately 30,000 hectares of agricultural land in the plain have been salted and left out of production and use. The deficiency of the drainage network in the farmlands was another important factor in the salinity of soil and groundwater. Moreover, surface irrigation with these waters (saline groundwater) on heavier textured soil of the area usually leads to building up of salinity and sodicity problems and thus unsustainable crop yields. Therefore, there is a need to adopt specialized and efficient methods of irrigation like micro-irrigation which can help in attaining the twin objectives of higher productivity and optimum use of water [24].

Figure 1. Location map of Harran Plain.

Figure 2. Salinization of agricultural lands in the area.

Salinization in the Harran plain first started with the groundwater wells drilled in Akçakale after 1970. These wells drilled by the State Hydraulic Works in Akçakale supplied water from the groundwater due to the cheap cost. Saline groundwater in shallow depth occurred due to insufficient drainage network and saline soils began to form over time (Fig. 2). The amount of salinity increased due to drainage problems after 1995 starting irrigated farming. The salinization on the South of Harran plain has reached serious dimensions due to insufficient underground and surface drainage. The area of saline soils will increase in the near future unless the necessary rehabilitation works are carried out.

3. Electrokinetic Remediation Methods

Soil salinity can be represented by the high electrical conductivity (EC, dS/m) of the soil. Electrokinetic (EK) remediation has considerable potential for the decontamination of saline soils with low permeability. EK remediation technology is an innovative, sustainable and low-cost method used for the stabilization and restoration of saline soils [25-27]. The major advantage of the method is that it is cost-effective for both in-situ and ex-situ treatment (Fig. 3).

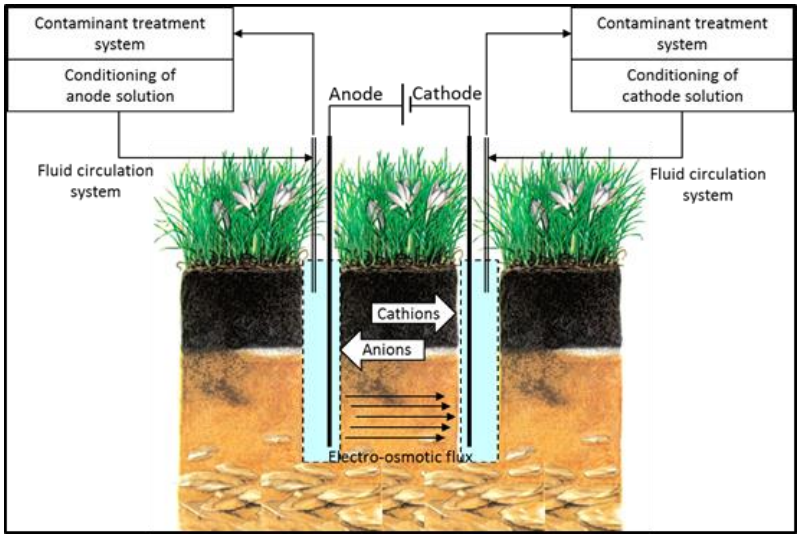

Figure 3. Application of the electrokinetic remediation in a contaminated site [28].

Soil salinity problems are very common in arid and semi-arid regions. Most of the cultivation activities of salt-affected soils are partly or totally unsuccessful. These failures are often due to the lack of appropriate diagnosis and the subsequent use of incorrect irrigation methods. This leads to the loss of both money and potential crop production. But the electrokinetic remediation process is the most promising, inexpensive and easily applicable green technology for the treatment of saline soils.

Electrokinetic treatment technologies basically work as a simple battery assembly. Accordingly, a series of electrodes (lead, copper, zinc, graphite, titanium, etc.) are connected to the contaminated zone and a certain amount of direct current is supplied to these electrodes. Due to the electrical current given to the electrical load in the ground a movement occurs towards the reverse charged electrode through the pollutants by electromigration, electro-osmosis, electrolysis, and diffusion. [29-31] (Fig. 4). The contaminant coming to the reverse charged electrode is deposited on the electrode, depending on its type and preference, or transported to the surface by the pump assembly and treated there. Recently, the EK remediation technique has been used for the recovery and reuse of saline soils. Tab.1. showed EK remediation studies in literature.

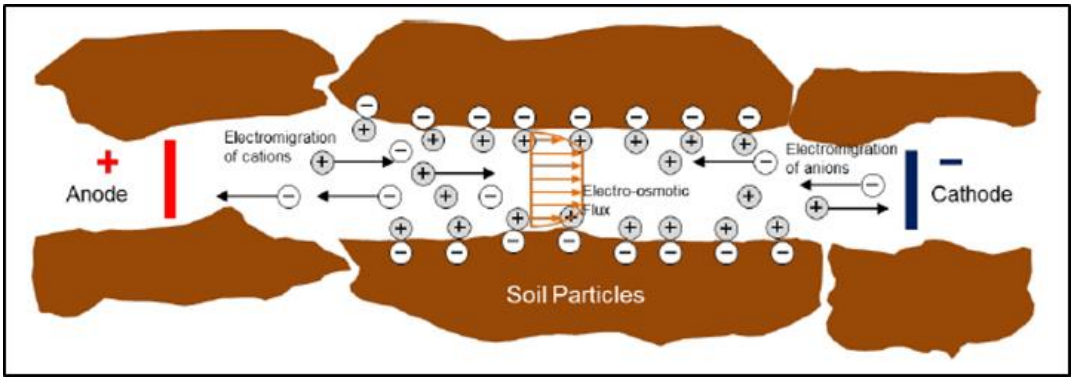

Figure 4. Transport mechanism of electrokinetic restoration [29].

Table 1. EK remediation studies in literatures.

Reference	Region	Efficiency/feasibility
[8]	Saline greenhouse soil at Gumi, Gyeongbuk, Republic of Korea. Surface soil (0-20 cm).	EK system could lower the corrosion rate of an electrode by approximately 38% and reduce electrical energy consumption by approximately 60% compared to the DC system, which would allow for longer application in the field.
[9]	An actual greenhouse in Gumi, Korea. Surface soil (0–20 cm).	The pulsed electrokinetic process lowered the electrical energy consumption to 42% of that of the conventional process
[16]	Surface saline soil (0–25 cm) was sampled from a greenhouse at Gumi, South Korea.	EK treatment resulted in the removal of approximately 30% of K and Ca from the system.
[32]	Dakhla Oasis, central Western Desert of Egypt. Surface soil (0-20 cm).	Linear spectral unmixing (LSU) proved to predict salinity more accurately with 76% correctness than mixture tuned matched filtering (MTMF) model (67%) and the band combination and spectral indices (55% at most).
[33]	North-west of Algeria, in the lower valleys of West Mostaganem, especially in the region of Ain Nouissy.	The proposed technique achieved removal rates of 83% and 58% for sodium and calcium ions respectively after 15 days of EK treatment.

4. Electrode Materials and Configuration

Fig. 5 shows a schematic of the EK remediation system. Soil compartments are placed between electrode chambers and are filled with compacted saline soil. Electrode compartments acted as overflow systems. The EK process involves direct application of a low voltage gradient or electrical current to polluted soil through installed electrodes as anode and cathode (graphite, platinum, etc). Salts, which have high water solubility and exist as charged ions, are transported and removed by electromigration (movement of charged ions), electro-osmosis (liquid flow from anode to the cathode through the pore spaces between particles), and electrophoresis (transfer of charged particles) from the soil surface.

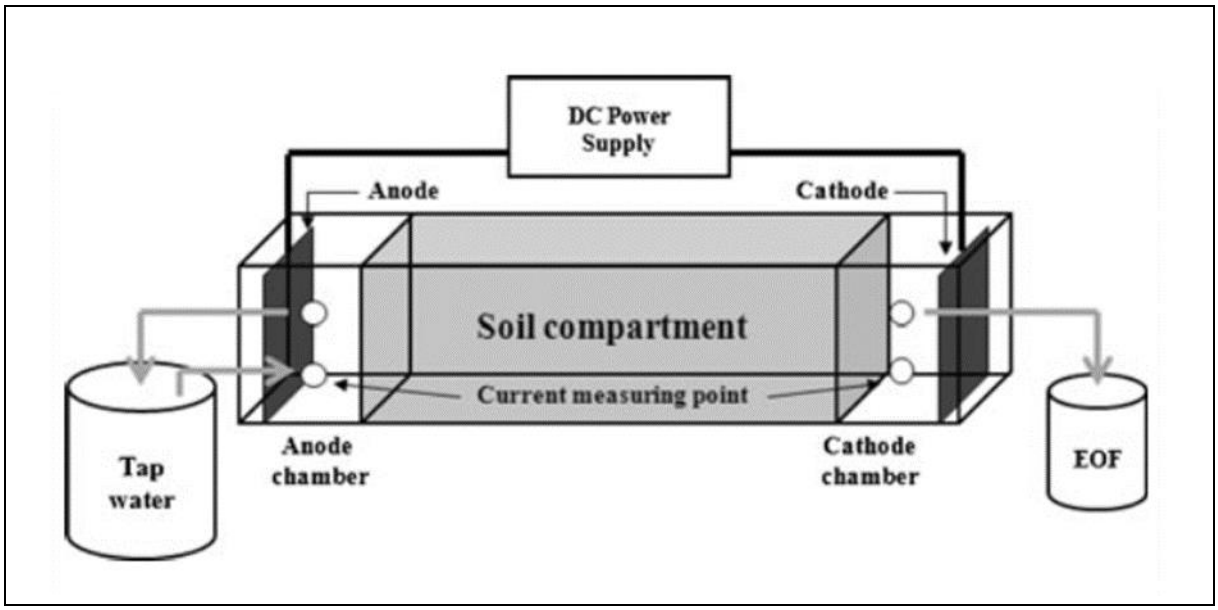

Figure 5. Schematic of an EK remediation system [7].

5. Applicability and Limitations of EK Remediation

Electrokinetic (EK) remediation technology has a significant potential for the cultivation of crops on saline soils (such as clay) with low permeability. This technology is an innovative, sustainable and inexpensive method proposed for the stabilization and reuse of soils in general and especially fine grains. EK remediation involves applying the electric current directly to the contaminated soil with a low voltage gradient or electrodes placed in the soil [33].

Long-term EK applications require a low voltage gradient because the property of the earth may be affected by the EK process under a high voltage gradient and may consume a lot of electricity [7]. In situ and ex situ applications of EK remediation have been developed [34]. The EK technique can be applied to widely dispersed pollutants in soils containing clay or sand in both unsaturated and saturated regions. The EC can remove polar and / or water-soluble organic compounds such as contaminants, heavy metals, arsenic, nitrates, phosphates, halogenides and cyanides, phenols and nitro aromatics (such as TNT).

6. Conclusion

As a result, the salinity problem is an important issue in the Harran Plain in Sanliurfa due to the high groundwater levels. In recent years, agricultural lands in this region have become impracticable and unusable. Electrokinetic treatment is an un-attempt, innovative and clean technology for the restoration of saline soils for this region. This process is thought to be the most effective, promising technique for the optimal and sustainable improvement of salt-affected soils in the Harran plain.

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Research Article

BLOW UP OF SOLUTIONS FOR A NONLINEAR VISCOELASTIC WAVE EQUATIONS WITH VARIABLE EXPONENTS

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Abstract: *The main purpose of this work is to study the blow up of solutions for the viscoelastic wave equation with variable exponents in a bounded domain. Our result extends the one in [11] to problems with variable exponent nonlinearities.*

Keywords: *Viscoelastic wave equation; Blow up; Variable exponent.*

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1. Introduction

In this work, we investigate the nonlinear viscoelastic wave equation

$$u_{tt}-\Delta u+\int_0^t g\left(t-\tau\right)\Delta u\left(\tau\right)d\tau+\left|u_t\right|^{p(x)-2}u_t=\left|u\right|^{q(x)-2}u,\quad (x,t)\in\Omega\times(0,T), \tag{1}$$

with the inital-boundary conditions

$$u\left(x,0\right)=u_0\left(x\right),\ u_t\left(x,0\right)=u_1\left(x\right),\quad x\in\Omega, \tag{2}$$

and

$$u\left(x,t\right)=0,\qquad x\in\partial\Omega, \tag{3}$$

here Ω is a regular and bounded domain in R^n $(n\geq 1)$ with smooth boundary $\partial\Omega$.

The variable exponents $p\left(.\right)$ and $q\left(.\right)$ are given as measurable functions on Ω satisfying

$$2\leq p^-\leq p\left(x\right)\leq p^+<q^-\leq q\left(x\right)\leq q^+\leq q^* \tag{4}$$

where

$$\begin{aligned} p^- &= ess\inf_{x\in\Omega} p(x), \quad p^+ = ess\sup_{x\in\Omega} p(x), \\ q^- &= ess\inf_{x\in\Omega} q(x), \quad q^+ = ess\sup_{x\in\Omega} q(x), \end{aligned}$$

and

$$q^* = \begin{cases} \infty, \text{ if } n = 1, 2, \\ \frac{2n}{n-2}, \text{ if } n \geq 3. \end{cases}$$

We make some assumptions on g :

(A1) Let $g \in C^1$ and

$$1 - \int_0^\infty g(\tau)\, d\tau = l > 0.$$

(A2) $g(\tau) \geq 0$, $g'(\tau) \leq 0$ and

$$\int_0^\infty g(\tau)\, d\tau < \frac{q^-(1-\xi) - 2}{q^-(1-\xi) - 2 + \frac{1}{4q^-(1-\xi)}}, \quad 0 < \xi < 1.$$

Remark 1 *There are some functions $g(\tau)$ satisfying (A1) and (A2). An example is*

$$g(\tau) = e^{-(\tau+1)}.$$

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When $p(x)$ and $q(x)$ are constants, (1) become the following the viscoelastic wave equation

$$u_{tt} - \Delta u + \int_0^t g(t-\tau)\, \Delta u(\tau)\, d\tau + |u_t|^{p-2} u_t = |u|^{q-2} u. \tag{5}$$

In [11], the author proved nonexistence of global solutions, for the equation (5). In [12], the same author extended this result in the case of positive initial energy. Later, some authors studied nonexistence of solutions of the equation (5) (see [17, 18]).

Without the viscoelastic term ($g = 0$) the problem (1) reduces to the following form

$$u_{tt} - \triangle u + |u_t|^{p(x)-2} u_t = |u|^{q(x)-2} u. \tag{6}$$

Messaoudi et al. [13] studied the local existence and nonexistence of the solutions of the equation (6). For more results concerning nonexistence of global solutions, see [2, 7, 8, 10]. For more results about the variable exponent spaces we refer the readers to [1, 14].

Motivated by the above results, in this work, we prove the blow up of solutions (1) under some conditions.

The outline of this work is as follows: In section 2, we state some results about the variable exponent Lebesgue spaces $L^{p(x)}(\Omega)$ and Sobolev spaces $W^{1,p(x)}(\Omega)$. In section 3, the blow up results will be proved.

2. Preliminaries

In this part, we state some results about the variable exponent Lebesgue spaces $L^{p(x)}(\Omega)$ and Sobolev spaces $W^{1,p(x)}(\Omega)$ (see [5, 6, 9, 15]).

Let $p:\Omega\to[1,\infty]$ be a measurable function, here Ω is a domain of R^n. We define the Lebesgue space with a variable exponent $p(.)$ by

$$L^{p(x)}(\Omega)=\left\{u:\Omega\to R,\ \text{measurable and}\ \int_{\Omega}|u|^{p(x)}\,dx<\infty\right\},$$

endowed with the Luxemburg norm

$$\|u\|_{p(x)}=\inf\left\{\lambda>0:\int_{\Omega}\left|\frac{u}{\lambda}\right|^{p(x)}dx\le 1\right\},$$

$L^{p(x)}(\Omega)$ is a Banach space.

The Sobolev space with a variable exponent is defined by

$$W^{1,p(x)}(\Omega)=\left\{u\in L^{p(x)}(\Omega):\nabla u\ \text{exists and}\ |\nabla u|\in L^{p(x)}(\Omega)\right\}.$$

Variable exponent Sobolev space is a Banach space with the following the norm

$$\|u\|_{1,p(x)}=\|u\|_{p(x)}+\|\nabla u\|_{p(x)}.$$

The space $W_0^{1,p(x)}(\Omega)$ is defined as the closure of $C_0^{\infty}(\Omega)$ in $W^{1,p(x)}(\Omega)$ with respect to the norm $\|u\|_{1,p(x)}$. For $u\in W_0^{1,p(x)}(\Omega)$, we can define an equivalent norm

$$\|u\|_{1,p(x)}=\|\nabla u\|_{p(x)}.$$

Let the variable exponent $p(.)$ satisfy the log-Hölder continuity condition:

$$|p(x)-p(y)|\le\frac{A}{\log\frac{1}{|x-y|}},\ \text{for all}\ x,y\in\Omega\ \text{with}\ |x-y|<\delta,\tag{7}$$

where $A>0$ and $0<\delta<1$.

Lemma 2 *[5] (Poincare inequality) Let Ω be a bounded domain of R^n and $p(.)$ satisfies log-Hölder condition, then*

$$\|u\|_{p(.)}\le c\|\nabla u\|_{p(.)},\ \textit{for all}\ u\in W_0^{1,p(.)}(\Omega),$$

where $c=c(p^-,p^+,|\Omega|)>0$.

Lemma 3 *[5] Let $p(.)\in C(\overline{\Omega})$ and $q:\Omega\to[1,\infty)$ be a measurable function and satisfy*

$$\operatorname*{essinf}_{x\in\overline{\Omega}}(p^*(x)-q(x))>0.$$

Then the Sobolev embedding $W_0^{1,p(.)}(\Omega)\hookrightarrow L^{q(.)}(\Omega)$ is continuous and compact. Where

$$p^*(x)=\begin{cases}\frac{np(x)}{\operatorname*{esssup}_{x\in\overline{\Omega}}(n-p(x))}, & \textit{if}\ p^-<n\\ \infty, & \textit{if}\ p^-\ge n.\end{cases}$$

The local existence of solutions for the problem (1) that can be established by combining arguments of [3, 7, 13].

Theorem 4 *(Local existence and uniqueness). Assume that (A1), (A2), (4) and (7) holds, and that* $(u_0, u_1) \in H_0^1(\Omega) \times L^2(\Omega)$. *Then problem (1) has a unuque local solution*

$$u \in C\left([0,T); H_0^1(\Omega)\right), \quad u_t \in C\left([0,T); L^2(\Omega)\right) \cap L^{p(.)}(\Omega \times (0,T)).$$

3. Blow up

In this part, we prove that the blow up of the solution for problem (1). Firstly, we give following lemmas:

Lemma 5 *[13] If* $q : \Omega \to [1, \infty)$ *is a measurable function and*

$$2 \leq q^- \leq q(x) \leq q^+ < \frac{2n}{n-2}; \quad n \geq 3 \tag{8}$$

holds. Then, we have following inequalities:

i)

$$\rho_{q(.)}^{\frac{s}{q^-}}(u) \leq c\left(\|\nabla u\|^2 + \rho_{q(.)}(u)\right), \tag{9}$$

ii)

$$\|u\|_{q^-}^s \leq c\left(\|\nabla u\|^2 + \|u\|_{q^-}^{q^-}\right), \tag{10}$$

iii)

$$\rho_{q(.)}^{\frac{s}{q^-}}(u) \leq c\left(|H(t)| + \|u_t\|^2 + \rho_{q(.)}(u)\right), \tag{11}$$

iv)

$$\|u\|_{q^-}^s \leq c\left(|H(t)| + \|u_t\|^2 + \|u\|_{q^-}^{q^-}\right), \tag{12}$$

v)

$$c\|u\|_{q^-}^{q^-} \leq \rho_{q(.)}(u) \tag{13}$$

for any $u \in H_0^1(\Omega)$ *and* $2 \leq s \leq q^-$. *Where* $\rho_{q(.)}(u) = \int_\Omega |u|^{q(.)}\,dx$, *and* $c > 1$ *a positive constant and*

$$H(t) = -\frac{1}{2}\|u_t\|^2 - \frac{1}{2}\left(1 - \int_0^t g(\tau)\,d\tau\right)\|\nabla u\|^2 - \frac{1}{2}(g \circ \nabla u)(t) + \int_\Omega \frac{1}{q(x)}|u|^{q(x)}\,dx.$$

Lemma 6 *Suppose that (A1), (A2), (4) and (7) hold. Then*

$$E(t) = \frac{1}{2}\|u_t\|^2 + \frac{1}{2}\left(1 - \int_0^t g(\tau)\,d\tau\right)\|\nabla u\|^2 + \frac{1}{2}(g \circ \nabla u)(t) - \int_\Omega \frac{1}{q(x)}|u|^{q(x)}\,dx \tag{14}$$

is a nonincreasing function and

$$\begin{aligned} E'(t) &= -\int_\Omega \frac{1}{p(x)}|u_t|^{p(x)}\,dx - \frac{1}{2}g(\tau)\int_\Omega |\nabla u(t)|^2\,dxd\tau \\ &\quad + \frac{1}{2}\int_0^t g'(t-\tau)\int_\Omega [\nabla u(\tau) - \nabla u(t)]^2\,dxd\tau. \end{aligned}$$

Proof. Multiplying u_t on two sides of the problem (1), and integrating by part, we get

$$\begin{aligned} & \frac{d}{dt}\left[\frac{1}{2}\left\|u_t\right\|^2+\frac{1}{2}\left\|\nabla u\right\|^2-\int\limits_{\Omega}\frac{1}{q\left(x\right)}\left|u\right|^{q(x)}dx\right] \\ & -\int\limits_0^t\int\limits_{\Omega}g\left(t-\tau\right)\nabla u\left(\tau\right)\nabla u_t\left(t\right)dxd\tau \\ = & -\int\limits_{\Omega}\left|u_t\right|^{p(x)}dx. \end{aligned} \tag{15}$$

Now, we estimate last term in the left hand side of (15), we obtain

$$\begin{aligned} & \int\limits_0^t\int\limits_{\Omega}g\left(t-\tau\right)\nabla u\left(\tau\right)\nabla u_t\left(t\right)dxd\tau \\ = & \int\limits_0^t g\left(t-\tau\right)\int\limits_{\Omega}\nabla u_t\left(t\right)\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)+\nabla u\left(t\right)\right]dxd\tau \\ = & \int\limits_0^t g\left(t-\tau\right)\int\limits_{\Omega}\nabla u_t\left(t\right)\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)\right]dxd\tau+\int\limits_0^t g\left(t-\tau\right)\int\limits_{\Omega}\nabla u_t\left(t\right)\nabla u\left(t\right)dxd\tau \\ = & -\frac{1}{2}\int\limits_0^t g\left(t-\tau\right)\frac{d}{dt}\left[\int\limits_{\Omega}\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)\right]^2dx\right]d\tau \\ & +\frac{1}{2}\int\limits_0^t g\left(\tau\right)\left[\frac{d}{dt}\int\limits_{\Omega}\left|\nabla u\left(t\right)\right|^2dx\right]d\tau \\ = & -\frac{1}{2}\frac{d}{dt}\left[\int\limits_0^t g\left(t-\tau\right)\int\limits_{\Omega}\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)\right]^2dxd\tau\right]+\frac{1}{2}\int\limits_0^t g'\left(t-\tau\right)\int\limits_{\Omega}\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)\right]^2dxd\tau \\ & +\frac{1}{2}\frac{d}{dt}\left[\int\limits_0^t g\left(\tau\right)\int\limits_{\Omega}\left|\nabla u\left(t\right)\right|^2dxd\tau\right]-\frac{1}{2}g\left(\tau\right)\int\limits_{\Omega}\left|\nabla u\left(t\right)\right|^2dxd\tau. \end{aligned} \tag{16}$$

Finally, inserting (16) into (15), we get

$$\begin{aligned} & \frac{d}{dt}\left[\frac{1}{2}\left\|u_t\right\|^2+\frac{1}{2}\left(1-\int\limits_0^t g\left(\tau\right)d\tau\right)\left\|\nabla u\right\|^2+\frac{1}{2}\left(g\circ\nabla u\right)\left(t\right)-\int\limits_{\Omega}\frac{1}{q\left(x\right)}\left|u\right|^{q(x)}dx\right] \\ = & -\int\limits_{\Omega}\frac{1}{p\left(x\right)}\left|u_t\right|^{p(x)}dx-\frac{1}{2}g\left(\tau\right)\int\limits_{\Omega}\left|\nabla u\left(t\right)\right|^2dxd\tau+\frac{1}{2}\int\limits_0^t g'\left(t-\tau\right)\int\limits_{\Omega}\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)\right]^2dxd\tau \\ \leq & \ 0, \end{aligned} \tag{17}$$

where

$$\left(g\circ\nabla u\right)\left(t\right)=\int\limits_0^t g\left(t-\tau\right)\int\limits_{\Omega}\left[\nabla u\left(\tau\right)-\nabla u\left(t\right)\right]^2dxd\tau.$$

■

Now, we state and prove our blow up result.

Theorem 7 *Under the assumptions of Theorem 4, and*

$$E(0) < 0.$$

Then the solution of the problem (1) blow up in finite time.

Proof. Let

$$H(t) = -E(t).$$

We see from (17) that $H(t) \geq 0$. Also, by the definition $H(t)$, we have

$$\begin{aligned} H(t) &= -\frac{1}{2}\|u_t\|^2 - \frac{1}{2}\left(1 - \int_0^t g(\tau)\,d\tau\right)\|\nabla u\|^2 - \frac{1}{2}(g \circ \nabla u)(t) + \int_\Omega \frac{1}{q(x)}|u|^{q(x)}\,dx \\ &\leq \int_\Omega \frac{1}{q(x)}|u|^{q(x)}\,dx \\ &\leq \frac{1}{q^-}\rho_{q(.)}(u). \end{aligned} \tag{18}$$

Now, we define $\Psi(t)$ as follows

$$\Psi(t) = H^{1-\sigma}(t) + \varepsilon \int_\Omega u u_t dx, \tag{19}$$

for ε small to be chosen later and

$$0 < \sigma \leq \min\left\{\frac{q^- - p^+}{(p^+ - 1)q^-}, \frac{q^- - 2}{2q^-}\right\}. \tag{20}$$

The time derivative of (19) and using Eq. (1), we have

$$\begin{aligned} \Psi'(t) &= (1-\sigma)H^{-\sigma}(t)H'(t) + \varepsilon\int_\Omega \left(u_t^2 + u u_{tt}\right)dx \\ &= (1-\sigma)H^{-\sigma}(t)H'(t) + \varepsilon\|u_t\|^2 - \varepsilon\|\nabla u\|^2 \\ &\quad + \varepsilon\int_0^t g(t-\tau)\int_\Omega \nabla u(t)\nabla u(\tau)\,dx d\tau \\ &\quad + \varepsilon\int_\Omega |u|^{q(.)}\,dx - \varepsilon\int_\Omega u u_t |u_t|^{p(.)-2}\,dx \\ &= (1-\sigma)H^{-\sigma}(t)H'(t) + \varepsilon\|u_t\|^2 - \varepsilon\|\nabla u\|^2 \\ &\quad + \varepsilon\int_\Omega |u|^{q(.)}\,dx - \varepsilon\int_\Omega u u_t |u_t|^{p(.)-2}\,dx \\ &\quad + \varepsilon\int_0^t g(\tau)\,d\tau\|\nabla u\|^2 + \varepsilon\int_0^t g(t-\tau)\int_\Omega \nabla u(t)\left[\nabla u(\tau) - \nabla u(t)\right]dx d\tau. \end{aligned} \tag{21}$$

By using Cauchy-Schwarz and Young's inequalities, we have

$$\begin{aligned}
&\int_0^t g(t-\tau)\int_\Omega \nabla u(t)\left[\nabla u(\tau)-\nabla u(t)\right]dxd\tau \\
\leq\ &\int_0^t g(t-\tau)\left\|\nabla u(t)\right\|\left\|\nabla u(\tau)-\nabla u(t)\right\|d\tau \\
\leq\ &\lambda(g\circ\nabla u)(t)+\frac{1}{4\lambda}\int_0^t g(\tau)\,d\tau\left\|\nabla u\right\|^2,\quad \lambda>0. \qquad (22)
\end{aligned}$$

Substituting (22) into (21), we have

$$\begin{aligned}
\Psi'(t) \geq\ &(1-\sigma)H^{-\sigma}(t)H'(t)+\varepsilon\left\|u_t\right\|^2-\varepsilon\left\|\nabla u\right\|^2 \\
&+\varepsilon\int_\Omega |u|^{q(.)}dx-\varepsilon\int_\Omega uu_t\left|u_t\right|^{p(.)-2}dx+\varepsilon\int_0^t g(\tau)\,d\tau\left\|\nabla u\right\|^2 \\
&-\varepsilon\lambda(g\circ\nabla u)(t)-\frac{\varepsilon}{4\lambda}\int_0^t g(\tau)\,d\tau\left\|\nabla u\right\|^2
\end{aligned}$$

By using the definition of the $H(t)$, we have

$$\begin{aligned}
-\varepsilon q^-(1-\xi)H(t) =\ &\frac{\varepsilon q^-(1-\xi)}{2}\left\|u_t\right\|^2+\frac{\varepsilon q^-(1-\xi)}{2}\left(1-\int_0^t g(\tau)\,d\tau\right)\left\|\nabla u\right\|^2 \\
&+\frac{\varepsilon q^-(1-\xi)}{2}(g\circ\nabla u)(t)-\varepsilon q^-(1-\xi)\int_\Omega \frac{1}{q(x)}|u|^{q(.)}dx, \qquad (23)
\end{aligned}$$

where $0<\xi<1$.

Subtracting and adding (23) on the right hand side of (21), we get

$$\begin{aligned}
\Psi'(t) \geq\ &(1-\sigma)H^{-\sigma}(t)H'(t)+\varepsilon q^-(1-\xi)H(t)+\varepsilon\left(\frac{q^-(1-\xi)}{2}+1\right)\left\|u_t\right\|^2 \\
&+\varepsilon\left[\frac{q^-(1-\xi)}{2}\left(1-\int_0^t g(\tau)\,d\tau\right)-1+\left(1-\frac{1}{4\lambda}\right)\int_0^t g(\tau)\,d\tau\right]\left\|\nabla u\right\|^2 \\
&\varepsilon\left(\frac{q^-(1-\xi)}{2}-\lambda\right)(g\circ\nabla u)(t)+\varepsilon\xi\int_\Omega |u|^{q(.)}dx-\varepsilon\int_\Omega uu_t\left|u_t\right|^{p(.)-2}dx. \qquad (24)
\end{aligned}$$

Then, for ξ small enough, we get

$$\begin{aligned}
\Psi'(t) \geq\ &\varepsilon\beta\left[H(t)+\left\|u_t\right\|^2+\left\|\nabla u\right\|^2+(g\circ\nabla u)(t)+\rho_{q(.)}(u)\right] \\
&+(1-\sigma)H^{-\sigma}(t)H'(t)-\varepsilon\int_\Omega uu_t\left|u_t\right|^{p(.)-2}dx \qquad (25)
\end{aligned}$$

where

$$\begin{aligned}
\beta =\ &\min\left\{q^-(1-\xi),\ \frac{q^-(1-\xi)}{2}-\lambda,\ \frac{q^-(1-\xi)}{2}+1,\ \varepsilon\xi,\right. \\
&\left.\frac{q^-(1-\xi)}{2}\left(1-\int_0^t g(\tau)\,d\tau\right)-1+\left(1-\frac{1}{4\lambda}\right)\int_0^t g(\tau)\,d\tau\right\} \\
>\ &0
\end{aligned}$$

and

$$\rho_{q(.)}(u) = \int_{\Omega} |u|^{q(.)}\, dx.$$

By using the following Young's inequality

$$XY \leq \frac{\delta^k X^k}{k} + \frac{\delta^{-l} Y^l}{l},$$

where $X, Y \geq 0,\ \delta > 0,\ k, l \in R^+$ such that $\frac{1}{k} + \frac{1}{l} = 1$. As a result, applying the previous we obtain

$$\begin{aligned}\int_{\Omega} u\,|u_t|^{p(.)-1}\, dx &\leq \int_{\Omega} \frac{1}{p(x)} \delta^{p(x)} |u|^{p(x)}\, dx + \int_{\Omega} \frac{p(x)-1}{p(x)} \delta^{-\frac{p(x)}{p(x)-1}} |u_t|^{p(x)}\, dx \\ &\leq \frac{1}{p^-} \int_{\Omega} \delta^{p(x)} |u|^{p(x)}\, dx + \frac{p^+ - 1}{p^+} \int_{\Omega} \delta^{-\frac{p(x)}{p(x)-1}} |u_t|^{p(x)}\, dx, \end{aligned} \tag{26}$$

where δ is constant depending on the time t and specified later. Inserting estimate (26) into (25), we get

$$\begin{aligned}\Psi'(t) &\geq \varepsilon\beta \left[H(t) + \|u_t\|^2 + \|\nabla u\|^2 + (g \circ \nabla u)(t) + \rho_{q(.)}(u)\right] \\ &\quad + (1-\sigma) H^{-\sigma}(t) H'(t) \\ &\quad - \varepsilon \frac{1}{p^-} \int_{\Omega} \delta^{p(x)} |u|^{p(x)}\, dx - \varepsilon \frac{p^+ - 1}{p^+} \int_{\Omega} \delta^{-\frac{p(x)}{p(x)-1}} |u_t|^{p(x)}\, dx. \end{aligned} \tag{27}$$

Let us choose δ, so that $\delta^{-\frac{p(x)}{p(x)-1}} = k_1 H^{-\sigma}(t)$, where $k_1 > 0$ is specified later, we obtain

$$\begin{aligned}\Psi'(t) &\geq \varepsilon\beta \left[H(t) + \|u_t\|^2 + \|\nabla u\|^2 + (g \circ \nabla u)(t) + \rho_{q(.)}(u)\right] \\ &\quad + (1-\sigma) H^{-\sigma}(t) H'(t) \\ &\quad - \varepsilon \frac{1}{p^-} \int_{\Omega} k^{1-p(x)} H^{\sigma(p(x)-1)}(t) |u|^{p(x)}\, dx - \varepsilon \frac{p^+ - 1}{p^+} \int_{\Omega} k H^{-\sigma}(t) |u_t|^{p(x)}\, dx \\ &\geq \varepsilon\beta \left[H(t) + \|u_t\|^2 + \|\nabla u\|^2 + (g \circ \nabla u)(t) + \rho_{q(.)}(u)\right] \\ &\quad + (1-\sigma) H^{-\sigma}(t) H'(t) \\ &\quad - \varepsilon \frac{k^{1-p^-}}{p^-} H^{\sigma(p^+-1)}(t) \int_{\Omega} |u|^{p(x)}\, dx - \varepsilon \left(\frac{p^+ - 1}{p^+}\right) k H^{-\sigma}(t) \int_{\Omega} |u_t|^{p(x)}\, dx \\ &\geq \varepsilon\beta \left[H(t) + \|u_t\|^2 + \|\nabla u\|^2 + (g \circ \nabla u)(t) + \rho_{q(.)}(u)\right] \\ &\quad + \left[(1-\sigma) - \varepsilon \left(\frac{p^+ - 1}{p^+}\right) k\right] H^{-\sigma}(t) H'(t) - \varepsilon \frac{k^{1-p^-}}{p^-} H^{\sigma(p^+-1)}(t) \int_{\Omega} |u|^{p(x)}\, dx. \end{aligned}$$

By using (13) and (18), we get

$$\begin{aligned} H^{\sigma\left(p^{+}-1\right)}(t)\int\limits_{\Omega}|u|^{p(x)}\,dx &\leq H^{\sigma\left(p^{+}-1\right)}(t)\left[\int\limits_{\Omega_{-}}|u|^{p^{-}}\,dx+\int\limits_{\Omega_{+}}|u|^{p^{+}}\,dx\right] \\ &\leq H^{\sigma\left(p^{+}-1\right)}(t)\,c\left[\left(\int\limits_{\Omega_{-}}|u|^{q^{-}}\,dx\right)^{\frac{p^{-}}{q^{-}}}+\left(\int\limits_{\Omega_{+}}|u|^{q^{-}}\,dx\right)^{\frac{p^{+}}{q^{-}}}\right] \\ &= H^{\sigma\left(p^{+}-1\right)}(t)\,c\left[\|u\|_{q^{-}}^{p^{-}}+\|u\|_{q^{-}}^{p^{+}}\right] \\ &\leq c\left(\frac{1}{q^{-}}\rho_{q(.)}(u)\right)^{\sigma\left(p^{+}-1\right)}\left[\left(\rho_{q(.)}(u)\right)^{\frac{p^{-}}{q^{-}}}+\left(\rho_{q(.)}(u)\right)^{\frac{p^{+}}{q^{-}}}\right] \\ &= c_{1}\left[\left(\rho_{q(.)}(u)\right)^{\frac{p^{-}}{q^{-}}+\sigma\left(p^{+}-1\right)}+\left(\rho_{q(.)}(u)\right)^{\frac{p^{+}}{q^{-}}+\sigma\left(p^{+}-1\right)}\right] \end{aligned} \tag{28}$$

where $\Omega_{-}=\{x\in\Omega:|u|<1\}$ and $\Omega_{+}=\{x\in\Omega:|u|\geq 1\}$.

We then use Lemma 5 and (20), for

$$s=p^{-}+\sigma q^{-}\left(p^{+}-1\right)\leq q^{-}$$

and

$$s=p^{+}+\sigma q^{-}\left(p^{+}-1\right)\leq q^{-},$$

to deduce, from (28),

$$H^{\sigma\left(p^{+}-1\right)}(t)\int\limits_{\Omega}|u|^{p(x)}\,dx\leq c_{1}\left[\|\nabla u\|^{2}+\rho_{q(.)}(u)\right]. \tag{29}$$

Thus, inserting estimate (29) into (25), we have

$$\begin{aligned} \Psi'(t) &\geq \varepsilon\left(\beta-\frac{k^{1-p^{-}}}{p^{-}}c_{1}\right)\left[H(t)+\|u_{t}\|^{2}+\|\nabla u\|^{2}+(g\circ\nabla u)(t)+\rho_{q(.)}(u)\right] \\ &\quad+\left[(1-\sigma)-\varepsilon\left(\frac{p^{+}-1}{p^{+}}\right)k\right]H^{-\sigma}(t)\,H'(t). \end{aligned} \tag{30}$$

Let us choose k large enough so that $\gamma=\beta-\frac{k^{1-p^{-}}}{p^{-}}c_{1}>0$, and picking ε small enough such that $(1-\sigma)-\varepsilon\left(\frac{p^{+}-1}{p^{+}}\right)k\geq 0$ and

$$\Psi(t)\geq\Psi(0)=H^{1-\sigma}(0)+\varepsilon\int_{\Omega}u_{0}u_{1}dx>0,\ \forall t\geq 0. \tag{31}$$

Consequently, (30) yields

$$\begin{aligned} \Psi'(t) &\geq \varepsilon\gamma\left[H(t)+\|u_{t}\|^{2}+\|\nabla u\|^{2}+(g\circ\nabla u)(t)+\rho_{q(.)}(u)\right] \\ &\geq \varepsilon\gamma\left[H(t)+\|u_{t}\|^{2}+\|\nabla u\|^{2}+(g\circ\nabla u)(t)+\|u\|_{q^{-}}^{q^{-}}\right], \end{aligned} \tag{32}$$

due to (13). Therefore we get

$$\Psi(t)\geq\Psi(0)>0,\ \text{for all } t\geq 0.$$

On the other hand, exploiting Hölder's inequality, we get

$$\begin{aligned}\left|\int_\Omega uu_t dx\right|^{\frac{1}{1-\sigma}} &\leq \|u\|^{\frac{1}{1-\sigma}} \|u_t\|^{\frac{1}{1-\sigma}} \\ &\leq C\left(\|u\|_{q^-}^{\frac{1}{1-\sigma}} \|u_t\|^{\frac{1}{1-\sigma}}\right).\end{aligned}$$

Young inequality gives

$$\left|\int_\Omega uu_t dx\right|^{\frac{1}{1-\sigma}} \leq C\left(\|u\|_{q^-}^{\frac{\mu}{1-\sigma}} + \|u_t\|^{\frac{\theta}{1-\sigma}}\right), \tag{33}$$

for $\frac{1}{\mu}+\frac{1}{\theta}=1$. We take $\theta = 2(1-\sigma)$, to obtain $\frac{\mu}{1-\sigma} = \frac{2}{1-2\sigma} \leq q^-$ by (20). Therefore, (33) becomes

$$\left|\int_\Omega uu_t dx\right|^{\frac{1}{1-\sigma}} \leq C\left(\|u_t\|^2 + \|u\|_{q^-}^s\right),$$

where $\frac{2}{1-2\sigma} \leq q^-$. By using (12), we get

$$\left|\int_\Omega uu_t dx\right|^{\frac{1}{1-\sigma}} \leq C\left(\|u_t\|^2 + \|u\|_{q^-}^{q^-} + H(t)\right).$$

Thus,

$$\begin{aligned}\Psi^{\frac{1}{1-\sigma}}(t) &= \left[H^{1-\sigma}(t) + \varepsilon\int_\Omega uu_t dx\right]^{\frac{1}{1-\sigma}} \\ &\leq 2^{\frac{\sigma}{1-\sigma}}\left(H(t) + \varepsilon^{\frac{1}{1-\sigma}}\left|\int_\Omega uu_t dx\right|^{\frac{1}{1-\sigma}}\right) \\ &\leq C\left(\|u_t\|^2 + \|u\|_{q^-}^{q^-} + H(t)\right) \\ &\leq C\left(H(t) + \|u_t\|^2 + \|\nabla u\|^2 + (g\circ\nabla u)(t) + \|u\|_{q^-}^{q^-}\right)\end{aligned} \tag{34}$$

where

$$(a+b)^p \leq 2^{p-1}(a^p + b^p)$$

is used. Consequently a combining of (32) and (34), for some $\xi > 0$, we have

$$\Psi'(t) \geq \xi\Psi^{\frac{1}{1-\sigma}}(t). \tag{35}$$

Integration of (35) over $(0,t)$ yield

$$\Psi^{\frac{\sigma}{1-\sigma}}(t) \geq \frac{1}{\Psi^{-\frac{\sigma}{1-\sigma}}(0) - \frac{\xi\sigma t}{1-\sigma}}.$$

Therefore $\Psi(t)$ blow up in a finite time

$$T^* \leq \frac{1-\sigma}{\xi\sigma\Psi^{\frac{\sigma}{1-\sigma}}(0)}.$$

Then, the proof is complete. ■

4. Conclusion

In this work, we obtained the blow up for a nonlinear viscoelastic wave equations with variable exponents in a bounded domain. This improves and extends many results in the literature.

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Research Article

EXISTENCE RESULTS FOR STEKLOV PROBLEM WITH NONLINEAR BOUNDARY CONDITION

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Abstract: *In this article, we study the nonlinear Steklov boundary value problem. The existence of a nontrivial weak solution is obtained on variable exponent Sobolev spaces, by means of the Mountain Pass theorem.*

Keywords: *p(x)-Laplace operator, variational methods, Steklov boundary value, Mountain Pass theorem.*

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1. Introduction

The purpose of this paper is to study the following Steklov problem involving the p(x)-Laplacian,

$$\begin{cases} -div\left(a(x)\left|\nabla u\right|^{p(x)-2}\nabla u\right)+\left|u\right|^{p(x)-2}u = f(x,u), x\in\Omega \\ a(x)\left|\nabla u\right|^{p(x)-2}\dfrac{\partial u}{\partial \upsilon} = \left|u\right|^{q(x)-2}u, x\in\partial\Omega \end{cases} \qquad (P)$$

where $\Omega\subset R^N$ $(N\geq 2)$ is a bounded with smooth boundary, p is continuous functions on $\overline{\Omega}$ such that $p^- := \inf\limits_{x\in\overline{\Omega}} p(x)>1$, q is continuous functions on $\partial\Omega$ such that $q^- := \inf\limits_{x\in\partial\Omega} q(x)>1$, and $p(x)\neq q(y)$ for any $x\in\overline{\Omega}, y\in\partial\Omega$, $\Delta_{p(x)}u := div\left(\left|\nabla u\right|^{p(x)-2}\nabla u\right)$ denotes the $p(x)-$Laplace operator, $f:\Omega xR\to R$ is a Carathéodory function, $\dfrac{\partial u}{\partial \upsilon}$ is the outer unit normal derivative on $\partial\Omega$ and $a(x)$ is a function which satisfies the conditions $0<a_1\leq a(x)\leq a_2$ where a_1 and a_2 are positive constants.

The study of differential equations and variational problems with $p(x)-$ growth conditions is a new and interesting topic in the last few years. The interest in studying such problems was stimulated by their application in mathematical physics, more precisely in elastic mechanics **[25]**, electrorheological fluids and stationary thermo-rheological viscous flows of non-Newtonian fluids, image processing [8,12,19,20**]** and the mathematical description of the processes filtration of an idea barotropic gas through a porous medium [3,7]. Many results have been obtained on this kind of problems, for instance, we here cite [1,4,10,13,14,16,18,21,22].

Problems of type (P) has been intensively studied by many authors [2,4,5,6,9,17]. In [24]**,** the authors investigated the existence and multiple results by using a variation of the Mountain Pass the

following $p(x)-$Laplacian with nonlinear boundary conditions in bounded domain Ω

$$\begin{cases} -div\left(a(x)\left|\nabla u\right|^{p(x)-2}\nabla u\right)+b(x)\left|u\right|^{p(x)-2}u=\lambda f(x,u), x\in\Omega \\ a(x)\left|\nabla u\right|^{p(x)-2}\dfrac{\partial u}{\partial \upsilon}=c(x)\left|u\right|^{g(x)-2}u+\eta g(x,u), x\in\partial\Omega \end{cases}$$

where f and g functions satisfies the Ambrosetti-Rabinowitz type condition. We also mention that the authors **[11]** studied de existence result for following class of Steklov boundary value problems involving $p(x)-$Laplacian

$$\begin{cases} -\Delta_{p(x)}u+a(x)\left|u\right|^{p(x)-2}u=f(x,u), x\in\Omega \\ \left|\nabla u\right|^{p(x)-2}\dfrac{\partial u}{\partial \upsilon}=g(x,u), x\in\partial\Omega \end{cases}$$

Using the variational method, under suitable conditions a, f and g, they obtained results on the existence of solutions.

This paper is organized as follows. In Section 2, we present some necessary preliminary knowledge on variable exponent Lebesgue and Sobolev spaces. In Section 3, using Mountain Pass theorem and the variational method we show the existence nontrivial weak solutions of problem (P).

2.Preliminaries

In this section, we recall in what follows some definitions and basic properties of variable exponent Lebesgue-Sobolev spaces $L^{p(x)}(\Omega), W^{1,p(x)}(\Omega)$ and $W_0^{1,p(x)}(\Omega)$ [22,16,15].

Set $C_+(\overline{\Omega})=\{p;p(x)\in C_+(\overline{\Omega}), \inf\ p(x)>1, \text{for all } x\in\overline{\Omega}\}$.

For any $p(x)\in C_+(\overline{\Omega})$, we denote

$$1<p^-:=\inf_{x\in\Omega}p(x)\le p^+:=\sup_{x\in\Omega}p(x)<\infty.$$

Define the variable exponent Lebesgue space $L^{p(x)}(\Omega)$ by

$$L^{p(x)}(\Omega)=\left\{u\middle|u:\Omega\to R \text{ is measurable, such that } \int_\Omega\left|u(x)\right|^{p(x)}dx<\infty\right\}$$

We define a norm, the so-called Luxemburg norm, on this space by the formula

$$\left|u\right|_{p(x)}:=\inf\left\{\lambda>0:\int_\Omega\left|\frac{u(x)}{\lambda}\right|^{p(x)}dx\le 1\right\}$$

and $\left(L^{p(x)}(\Omega),\left|u\right|_{p(x)}\right)$ becomes a Banach space.

Let $a:\partial\Omega\to R$ be measurable. Define the weighted variable exponent Lebesgue space

$$L^{p(x)}_{a(x)}(\partial\Omega)=\left\{u\middle|u:\partial\Omega\to R \text{ is measurable and} \int_{\partial\Omega}\left|a(x)\right|\left|u\right|^{p(x)}dx<+\infty\right\},$$

With the norm

$$\left|u\right|_{(p(x),a(x))}=\inf\left\{\kappa>0;\int_{\partial\Omega}\left|a(x)\right|\left|\frac{u}{\kappa}\right|^{p(x)}d\sigma\le 1\right\}$$

Where $d\sigma$ is the measure on the boundary. Then $L^{p(x)}_{a(x)}(\partial\Omega)$ is a Banach space. In particular, $a \in L^{\infty}(\partial\Omega), L^{p(x)}_{a(x)}(\partial\Omega) = L^{p(x)}(\partial\Omega)$.

Proposition 2.1[14,21] *If* $p(x) \in L^{\infty}$, *the conjugate space* $L^{p(x)}(\Omega)$ *is* $L^{p'(x)}(\Omega)$ *where* $\frac{1}{p(x)} + \frac{1}{p'(x)} = 1$. *For any* $u \in L^{p(x)}(\Omega)$ *and* $v \in L^{p'(x)}(\Omega)$, *we have*

$$\left|\int_{\Omega} uv dx\right| \le \left(\frac{1}{p^{-}} + \frac{1}{p^{+}}\right)|u|_{p(x)}|v|_{p'(x)}.$$

The modular of the $L^{p(x)}(\Omega)$ space, which is the mapping $\rho_{p(x)} : L^{p(x)}(\Omega) \to R$ defined by

$$\rho_{p(x)}(u) = \int_{\Omega} |u|^{p(x)} dx, \quad \forall u \in L^{p(x)}(\Omega).$$

Proposition 2.2 [15,24] *If* $u, u_n \in L^{p(x)}(\Omega)$ *(n=1,2,...) and* $p^{+} < \infty$, *we have*

(i) $|u|_{p(x)} < 1 (= 1, > 1) \Leftrightarrow \rho_{p(x)}(u) < 1 (= 1, > 1)$,

(ii) $\min\left(|u|^{p^{-}}_{p(x)}, |u|^{p^{+}}_{p(x)}\right) \le \rho_{p(x)}(u) \le \max\left(|u|^{p^{-}}_{p(x)}, |u|^{p^{+}}_{p(x)}\right)$,

(iii) $|u_n - u|_{p(x)} \to 0 (\to \infty) \Leftrightarrow \rho_{(p(x),\Omega)}(u_n - u) \to 0 (\to \infty)$.

Proposition 2.3 [24] *Denote* $\rho_{p(x)}(u) = \int_{\partial\Omega} |u|^{p(x)} d\sigma, \quad \forall u \in L^{p(x)}(\partial\Omega)$. *Then*

(i) $|u|_{L^{p(x)}(\partial\Omega)} \ge 1 \Leftrightarrow |u|^{p^{-}}_{L^{p(x)}(\partial\Omega)} \le \rho_{L^{p(x)}(\partial\Omega)}(u) \le |u|^{p^{+}}_{L^{p(x)}(\partial\Omega)}$,

(ii) $|u|_{L^{p(x)}(\partial\Omega)} < 1 \Leftrightarrow |u|^{p^{+}}_{L^{p(x)}(\partial\Omega)} \le \rho_{L^{p(x)}(\partial\Omega)}(u) \le |u|^{p^{-}}_{L^{p(x)}(\partial\Omega)}$.

Remark 2.4. It is noted that since $L^{p(x)}(\Omega) \to L^{1}_{loc}(\Omega)$ i.e., for any compact subset $K \subset \Omega$ there exists a constant $C_K > 0$ such that $\| fX_K \|_1 \le C_K \| f \|$. So every function in $L^{p(x)}(\Omega)$ has a distributional (weak) derivative, and variable exponent Sobolev space is well defined on $L^{p(x)}(\Omega)$.

The variable exponent Sobolev space $W^{1,p(x)}(\Omega)$ is denined by

$$W^{1,p(x)}(\Omega) = \left\{u \in L^{p(x)}(\Omega) : |\nabla u| \in L^{p(x)}(\Omega)\right\}$$

and equipped with the norm,

$$\|u\| := \inf\left\{\lambda > 0 : \int_{\Omega}\left|\frac{\nabla u(x)}{\lambda}\right|^{p(x)} + \int_{\Omega}\left|\frac{u(x)}{\lambda}\right|^{p(x)} dx \le 1\ \right\}.$$

For $u \in W^{1,p(x)}(\Omega)$, if we define

$$\|u\|_a := \inf\left\{\lambda > 0 : \int_{\Omega} a(x)\left|\frac{\nabla u(x)}{\lambda}\right|^{p(x)} + \int_{\Omega} b(x)\left|\frac{u(x)}{\lambda}\right|^{p(x)} dx \le 1\ \right\}.$$

In view of assumptions $a(x)$ of and $b(x)$ ($b(x)$ is a function which satisfies the conditions $0 < b_1 \le b(x) \le b_2$ where b_1 and b_2 are positive constants), it is easy to see that $\|u\|_a$ is an equivalent norm on $W^{1,p(x)}(\Omega)$.

Proposition 2.5 [24] *Denote* $\Gamma(u) = \int_{\partial\Omega}\left(a(x)|\nabla u|^{p(x)} + |u|^{p(x)}\right) d\sigma, \quad \forall u \in W^{1,p(x)}(\partial\Omega)$. *Then*

(i) $\Gamma(u) \geq 1 \Rightarrow \varepsilon_1 \|u\|^{p^-} \leq \Gamma(u) \leq \varepsilon_2 \|u\|^{p^+}$

(ii) $\Gamma(u) \leq 1 \Rightarrow \varepsilon_3 \|u\|^{p^+} \leq \Gamma(u) \leq \varepsilon_4 \|u\|^{p^-}$

where $\varepsilon_1, \varepsilon_2, \varepsilon_3$ and ε_4 are positive constants independent of u.

Space $W_0^{1,p(x)}(\Omega)$ is denoted as the closure of $C_0^\infty(\Omega)$ in $W^{1,p(x)}(\Omega)$ with respect to the norm $|u|_{1,p(x)}$. For $u \in W_0^{1,p(x)}(\Omega)$, we can define an equivalent norm $\|u\| = |\nabla u|_{p(x)}$. Since Poincaré inequality holds [16], i.e. there exists a positive constant $C > 0$ such that

$$\|u\| \leq C|\nabla u|_{p(x)} \text{ for all } u \in W_0^{1,p(x)}(\Omega).$$

Proposition 2.6 [16,24]

(i) *If* $1 < p^- \leq p^+ < \infty$ *then the spaces* $L^{p(x)}(\Omega)$, $W^{1,p(x)}(\Omega)$ *and* $W_0^{1,p(x)}(\Omega)$ *are separable, reflexive and uniformly convex Banach spaces,*

(ii) *If* $q(x) \in C_+(\overline{\Omega})$ *and* $q(x) < p^*(x)$ *for all* $x \in \overline{\Omega}$ *then the embedding* $W^{1,p(x)}(\Omega) \to L^{q(x)}(\Omega)$ *is compact and continuous,*

(iii) *If* $q(x) \in C_+(\partial\Omega)$ $q(x) < p^\partial(x)$ *and for all* $x \in \partial\Omega$ *then the trace embedding* $W^{1,p(x)}(\Omega) \to L^{q(x)}(\partial\Omega)$ *is compact and continuous.*

We define,

$$p^*(x) = \begin{cases} \dfrac{Np(x)}{N - p(x)}, & if \quad N > p(x) \\ \infty, & if \quad N \leq p(x) \end{cases} \quad \text{and} \quad p^\partial(x) = \begin{cases} \dfrac{(N-1)p(x)}{N - p(x)}, & if \quad N > p(x) \\ \infty, & if \quad N \leq p(x). \end{cases}$$

Definition 2.7 [23] *Let* X *be Banach spaces and the function* $I \in C^1(X, R)$. *We say that* I *satisfies Palais-Smale condition* ***(PS)*** *in* X *if any sequence* $\{u_n\}$ *in* X *such that* $I(u_n)$ *is bounded and* $I'(u_n) \to 0$ *in* X^* *as* $n \to \infty$ *has a convergent subsequence.*

Lemma 2.8 8 (Mountain Pass Theorem) [23] *Let* X *be a Banach space and the function* $I \in C^1(X, R)$ *satisfies Palais-Smale condition. Assume that* $I(0) = 0$ *and*

(i) *There exist two positive real numbers* η *and* r *such that* $I(u) \geq r$ *with* $\|u\| = r$,

(ii) *There exists* $u_1 \in X$ *such that* $\|u_1\| > r$, *and* $I(u) < 0$.

Put $G = \{\varphi \in C([0,1], X) : \varphi(0) = 0, \varphi(1) = u_1\}$. *Set* $\beta = \inf\{\max \varphi([0,1]) : \varphi \in G\}$. *Then* $\beta \geq r$ *and* β *is a critical value of* I.

Throughout this paper, the following hypotheses are assumed.

(f1) $f : \Omega x R \to R$ is Carathéodory condition such that

$$|f(x,t)| \leq c_1 + c_2|t|^{\alpha(x)-1}, \quad \forall (x,t) \in \Omega x R,$$

where $c_1, c_2 > 0$, $\alpha(x) \in C_+(\overline{\Omega})$ and $\alpha(x) < p^*(x)$,

(f2) $f(x,t)=o\left(|t|^{p^+-1}\right), t\to 0$; for all $x\in\Omega$,

(**AR):** Ambrosetti-Rabinowitz's condition; there exist $t^*>0$ and $\theta>p^+$ such that $0<\theta F(x,t)\le f(x,t)t,\ |t|\ge t^*$, for all $x\in\Omega$

where $F(x,t)=\int_0^t f(x,s)ds$ and $q^-\le q(x)<p^\partial(x)$ for all $q(x)\in C(\partial\Omega)$.

Theorem 2.9. *Assume that conditions* ***(f1), (f2), (AR),*** $q^->\theta,\alpha^-$, p^+ and $\alpha^->p^+$ *are satisfied, then problem* (P) *has at least one nontrivial weak solution.*

3. Main Results

Let X denote the variable exponent Sobolev space $W^{1,p(x)}(\Omega)$. We say that $u\in X/\{0\}$ is a weak solution of **(P)** if

$$\int_\Omega a(x)|\nabla u|^{p(x)-2}\nabla u\nabla v dx+\int_\Omega |u|^{p(x)-2}uvdx-\int_{\partial\Omega}|u|^{q(x)-2}uvd\sigma-\int_\Omega f(x,u)vdx=0$$

for all $v\in X$.

The energy functional corresponding to the problem (P) is defined as $I=X\to R$

$$I(u)=\int_\Omega \frac{a(x)|\nabla u|^{p(x)}+|u|^{p(x)}}{p(x)}-\int_{\partial\Omega}\frac{|u|^{q(x)}}{q(x)}d\sigma-\int_\Omega F(x,u)dx$$

where $F(x,u)=\int_0^u f(x,s)ds$ and $d\sigma$ is the measure on the boundary.

Proposition 3.1 [24] *If one denotes*

$$J(u)=\int_\Omega \frac{a(x)|\nabla u|^{p(x)}+|u|^{p(x)}}{p(x)}dx,\ \ \forall x\in X.$$

Then $J\in C^1(X,R)$ *and the derivative operator of* J *, denoted by* J' *, is*

$$\langle J'(u),v\rangle=\int_\Omega a(x)|\nabla u|^{p(x)-2}\nabla u\nabla v dx+\int_\Omega |u|^{p(x)-2}uvdx,\quad \forall u,v\in X,$$

and one has:

(i) $J':X\to X^*$ *is a continuous, bounded, and strictly monotone operator,*

(ii) J' *is a mapping of* (S^+) *type, that is, if* $u_n\to u$ *(weak convergent) in* X *and* $\limsup_{n\to\infty}\langle J'(u_n)-J'(u),u_n-u\rangle\le 0$ *, then* $u_n\to u$ *(strongly convergent) in* X *,*

(iii) $J':X\to X^*$ *is a homeomorphism.*

Proposition 3.2 [5,24] *If one denotes*

$$\varphi(u)=\int_{\partial\Omega}\frac{|u|^{q(x)}}{q(x)}d\sigma,\forall u\in X,$$

where $q(x)\in C_+(\partial\Omega)$ and $q(x)<p*(x)$ for any $x\in\partial\Omega$, then $\varphi\in C^1(X,R)$ *and the derivative operator of* φ *, denoted by* φ' *, is*

$$\langle \varphi'(u), v \rangle = \int_{\partial\Omega} |u|^{q(x)-2} uv d\sigma, \quad \forall u, v \in X,$$

and one has $\varphi : X \to R$ *and* $\varphi' : X \to X^*$ *are sequentially weak- strongly continuous, bounded, namely,* $u_n \to u$ *(weakly continuous) implies* $\varphi'(u_n) \to \varphi'(u)$ *(strongly continuous)* .

Therefore, from the assumption **(f1)**, Proposition 2.6, Proposition 3.1 and Proposition 3.2, it is easy to see that $I(u) \in C^1(X, R)$ and the critical points I are weak solutions of (P). Moreover, the derivate of I is the mapping $I' : X \to X^*$

$$\langle I'(u), v \rangle = \int_\Omega \left(a(x) |\nabla u|^{p(x)-2} \nabla u \nabla v + |u|^{p(x)-2} uv \right) dx - \int_{\partial\Omega} |u|^{p(x)-2} uv d\sigma - \int_\Omega f(x,u) v dx$$

for any $u, v \in X$ **[6].**

Lemma 3.3 *Suppose that* ***(f1), (f2), (AR)*** and $q^- > \theta$ *are satisfied, then* I *satisfies the* ***(PS)*** *condition.*

Proof. Assume that $\{u_n\} \subset X$ is a sequence which satisfies the properties:

$$I(u_n) \to C \text{ and } I'(u_n) \to 0 \text{ in } X^* \text{ as } n \to \infty, \tag{3.1}$$

where X^* is dual space of X and C is a positive constant. We prove that $\{u_n\}$ possesses a convergent subsequence. First, we show that $\{u_n\}$ is bounded in X. We assume by contradiction $\|u_n\| \to \infty$ as $n \to \infty$. Using ***(AR)*** $q^- > \theta$, (3.1), Proposition 2.2, Proposition 2.3 and considering $\|u_n\| > 1$, for n large enough, we can write

$$\begin{aligned}
&C + \|u_n\| \geq I(u_n) - \frac{1}{\theta} \langle I'(u_n), u_n \rangle \\
&= \int_\Omega \frac{1}{p(x)} \left(a(x) |\nabla u_n|^{p(x)} + |u_n|^{p(x)} \right) dx - \int_{\partial\Omega} \frac{1}{q(x)} |u_n|^{q(x)} d\sigma - \int_\Omega F(x, u_n) dx \\
&- \frac{1}{\theta} \left(\int_\Omega \left(a(x) |\nabla u_n|^{p(x)} + |u_n|^{p(x)} \right) dx - \int_{\partial\Omega} |u_n|^{q(x)} d\sigma - \int_\Omega f(x, u_n) u_u dx \right) \\
&\geq \left(\frac{\varepsilon_1}{p^+} - \frac{1}{\theta} \right) \|u_n\|^{p^-}
\end{aligned}$$

where $c_3 > 0$ is constant. Since $\theta > p^+$ we obtain that $\{u_n\}$ is bounded in X. Next, we show the strong converges to u_n X in. Since it $\{u_n\}$ is bounded in X, there exists u in X such that, up to a subsequence, u_n converges weakly to u in X . Taking into account (3.1), we have $\langle I'(u_u), u_n - u \rangle \to 0$. So, we have

$$\begin{aligned}
&\langle I'(u_n), u_n - u \rangle \\
&= \int_\Omega \left(a(x) |\nabla u_n|^{p(x)-2} \nabla u_n (\nabla u_n - \nabla u) + |u_n|^{p(x)-2} u_n (u_n - u) \right) dx \\
&- \int_{\partial\Omega} |u_n|^{q(x)-2} u_n (u_n - u) d\sigma - \int_\Omega f(x, u_n)(u_n - u) dx.
\end{aligned}$$

Using **(f1)** and Proposition 2.1, it follows

$$\left|\int_\Omega f(x,u_n)(u_n-u)dx\right| \le \left|\int_\Omega \left(c_1+c_2|u_n|^{\alpha(x)-1}\right)(u_n-u)dx\right|$$
$$\le c_1\int_\Omega |u_n-u|dx + c_4\left\||u_n|^{\alpha(x)-1}\right\|_{\alpha'(x)}|u_n-u|_{\alpha(x)}$$

where $c_4>0$ is constant. Because $\alpha(x)<p^*(x)$ (Proposition 2.6 (ii)), there exists u such that u_n converges weakly to u in X. Thanks to the compact embedding $X\to L^{\alpha(x)}(\Omega)$, we get

$$u_n \to u(strongly) \text{ in } L^{\alpha(x)}$$
$$u_n \to u \; a.e. x\in\Omega$$

So,

$$\int_\Omega f(x,u_n)(u_n-u)dx\to 0.$$

Similarly, by Proposition 2.1, Proposition 2.6 (iii) and Proposition 3.2, we have

$$\int_{\partial\Omega}|u_n|^{q(x)-2}u_n(u_n-u)d\sigma\to 0.$$

Thus,

$$\int_\Omega \left(a(x)|\nabla u_n|^{p(x)-2}\nabla u_n(\nabla u_n-\nabla u)+|u_n|^{p(x)-2}u_n(u_n-u)\right)dx\to 0.$$

Finally, from Proposition 3.1**,** we deduce that u_n converges strongly to u X in. Therefore, I it satisfies the (PS) condition.

Lemma 3.4 *Assume that conditions* ***(f1), (f2)****,* $q^->p^+,\alpha^-$ and $\alpha^->p^+$ *are fulfilled. Then, there exist two positive real numbers* ρ *and* r *such that* $I(u)\ge r$ *with* $\|u\|=\rho$.

Proof: For $\|u\|<1,$ by Proposition 2.5, we have

$$I(u)\ge \frac{\varepsilon_3}{p^+}\|u\|^{p^+}-\frac{1}{q^-}\int_{\partial\Omega}|u|^{q(x)}d\sigma-\int_\Omega F(x,t)dx$$

where c_7 is constant. Since we have the continuous embeddings $X\to L^{q(x)}(\partial\Omega)$ $X\to L^{p^+}(\Omega)$, and $X\to L^{\alpha(x)}(\Omega)$ (Proposition 2.6), there exist c_5, c_6 and c_7 positive constants such that for all $u\in X$

$$|u|_{q(x),\partial\Omega}\le c_5\|u\|,\quad |u|^{p^+}\le c_6\|u\| \text{ and } |u|_{\alpha(x),\Omega}\le c_8\|u\|. \tag{3.2}$$

Choose $\varepsilon>0$ small enough such that $\left(\varepsilon c_6^{p^+}+c_\varepsilon c_7^{\alpha^-}\right)<\frac{\varepsilon_4}{2p^+}$. Using **(f1)** and **(f2),** we have

$$|F(x,t)|\le \varepsilon|t|^{p^+}+c_\varepsilon|t|^{\alpha(x)}, \text{ for all } (x,t)\in\Omega\times\mathrm{R}. \tag{3.3}$$

Thus, using (3.2) and (3.3) for $\|u\|<1,$ we get

$$I(u)\ge \frac{\varepsilon_4}{p^+}\|u\|^{p^+}-\frac{c_5}{q^-}\|u\|^{q^-}-\varepsilon c_6^{p^+}\|u\|^{p^+}-c_\varepsilon c_7^{\alpha^-}\|u\|^{\alpha^-}$$
$$\ge \frac{\varepsilon_4}{p^+}\|u\|^{p^+}-\frac{c_5}{q^-}\|u\|^{q^-}-\varepsilon c_6^{p^+}\|u\|^{p^+}-c_\varepsilon c_7^{\alpha^-}\|u\|^{p^+}.$$
$$\ge \frac{\varepsilon_4}{2p^+}\|u\|^{p^+}-\frac{c_5}{q^-}\|u\|^{q^-}$$

Choose $c_5 \le \frac{q^- \varepsilon_4}{2p^+}$. It follows that there exist $r > 0$ s and $\rho > 0$ such that $I(u) \ge r$ with $\|u\| = \rho$. The proof of Lemma 3.4 is completed.

Lemma 3.5 *If* ***(f1), (f2)*** *and* ***(AR)*** *hold, there exists* $\phi \in X$ *such that* $\|\phi\| > \eta$ *and* $I(t\phi) < 0$ *for* $t > 0$.

Proof. Thanks to **(AR),** we obtain $|F(x,t)| \ge c_8 |t|^\theta$ for all $(x,t) \in \Omega \times R$. Moreover, when $t > 1$ is large enough, from Proposition 2.2, we obtain that

$$I(t\phi) = \int_\Omega \frac{a(x)|\nabla t\phi|^{p(x)} + |t\phi|^{p(x)}}{p(x)} dx - \int_{\partial\Omega} \frac{|t\phi|^{q(x)}}{q(x)} d\sigma - \int_\Omega F(x, t\phi) dx$$

$$\le \frac{t^{p^+}}{p^-} \int_\Omega \left(a(x)|\nabla \phi|^{p(x)} + |\phi|^{p(x)} \right) dx - \frac{t^{q^-}}{q^+} \int_{\partial\Omega} |\phi|^{q(x)} d\sigma - c_8 t^\theta \int_\Omega |\phi| dx$$

Since $\theta > p^+$ we conclude that $I(t\phi) \to -\infty$ $t \to \infty$ as. The proof is completed.

Proof of Theorem 2.9. From Lemma 3.3, Lemma 3.4, Lemma 3.5 and $I(0) = 0$, I satisfies all statements of Lemma 2.8. Therefore, I has at least one nontrivial critical point, i.e., problem ***(P)*** has a nontrivial weak solution. The proof is completed.

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Review

ROLE OF OXIDATIVE STRESS IN BIOLOGICAL SYSTEMS

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Abstract: *Oxidative stress is a phenomenon wherein there is an imbalance between the rate of oxidant formation and its elimination from the body. It has been known to be a promoting factor of various acute and chronic diseases, some of which are lethal. Oxidative stress usually occurs when the generation of oxidants, as a byproduct of the metabolic processes is much higher than usual. However, several exogenous sources such as pollution and alcohol have been known to be major factors in oxidative stress. Although the human body produces several antioxidants their inadequacy can be combatted by the consumption of food rich in antioxidants. The following review briefly highlights the generation of free radicals in the body, their effect on biomolecules and the role of oxidative stress in the human body.*

Keywords: *antioxidants, glycoxidative damage, Oxidative stress, reactive oxygen species (ROS), reactive nitrogen species (RNS)*

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1. Introduction

Oxygen is the most vital element required for life. The process of oxidation-reduction during metabolism generates free radicals or oxidants. The balance between the rate of oxidant formation and their elimination is essential for the systematic functioning of biological processes [1]. An imbalance in this rate may be caused due to a certain disturbance in the endogenous system or exogenous factors such as unhealthy diet, smoking, medicinal side-effects, etc. This phenomenon of imbalance has been called oxidative stress [2].

Oxidants can be grouped as radicals and non-radicals. Both reactive oxygen species (ROS), as well as reactive nitrogen species (RNS), can exist in radical and non-radical form. Radicals can exist independently and contain one or more unpaired electron in the valence shell. Superoxide ($O_2^{\cdot-}$), Peroxyl radical ($ROO^{\cdot}$), Alkoxy radicals ($RO\cdot$), Nitric oxide (nitrogen monoxide) ($NO^{\cdot}$), Nitrogen dioxide ($NO\cdot_2$) and Hydroxyl (-OH) ions are some of the examples of radical oxidants [2, 3]. They are highly reactive and attain stability either by donating or accepting electrons, whereas non-radical oxidants are lesser reactive but participate in free radical reactions. Nonradical oxidants include Hydrogen peroxide (H_2O_2), Hypochlorous acid (HOCl), Ozone (O_3), Singlet oxygen ($^1O_2$), Nitrous acid (HNO_2), Organic peroxides (ROOH), Aldehydes (HCOR), Peroxynitrite (ONOOH), etc [1,3].

2. Generation of Free Radicals

Free radicals may be generated inside the body, i.e. endogenously as a by-product of aerobic metabolism while exogenous sources can be related to the individual's lifestyle and environment (Fig.1.). During normal metabolism, each cell produces approximately 20 billion oxidants per day. The insufficient reduction of oxygen in mitochondria during the electron transport chain (ETC) can lead to the formation of hydrogen peroxide and hydroxyl radicals. As a self-defense mechanism, white blood cells produce nitric oxide (NO), superoxides and H_2O_2 to combat with the pathogenic microorganisms. Moreover, during the degradation of fatty acids, peroxisomes may produce hydrogen peroxide as by-products. Usually, this hydrogen peroxide is degraded in the cell by catalase. However, under certain conditions, it might escape the catalysis and contribute to oxidative stress. Furthermore, cytochrome P450 enzymes produce oxidant as a defense against ingested toxic chemicals [4].

The exogenous sources may include inhalation of free radicals present in the environment from ionizing radiations (Ultraviolet light), automobile exhaust (mainly ozone and nitrous oxide), burning of certain substances, etc. These factors have been known to lower the level of antioxidants in the body thereby contributing to oxidative stress [4]. Exposure to air pollution or smoke generates oxygen radicals while breathing. Cigarette smoking (active or passive), consumption of alcohol and unsaturated fat may put at risk the natural antioxidant system in the body thereby contributing to oxidative stress [1, 4].

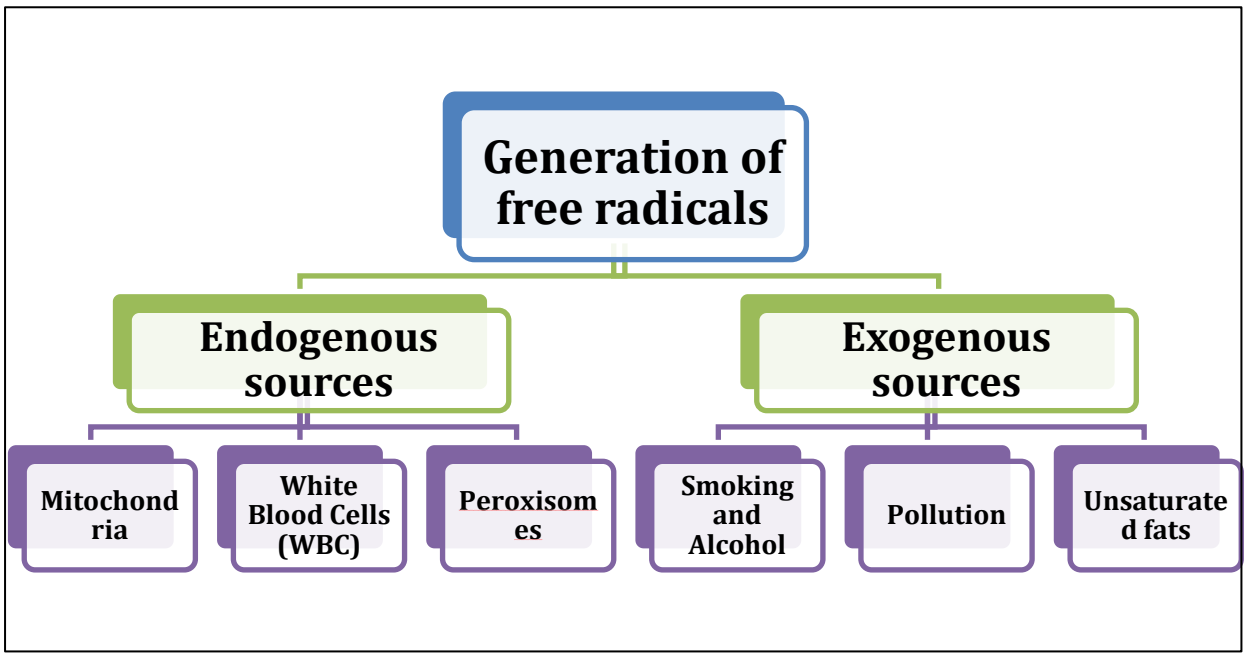

Figure 1. Sources for the generation of free radicals in the human body

3. Mechanism of action of free radicals

Most of the biomolecules and biochemical processes are affected by free radicals. However, the three major classes of biomolecules, i.e. nucleic acids, proteins, and lipids are severely affected by oxidative stress. These oxidized biomolecules may independently or simultaneously contribute to diseases affecting human health [5].

3.1. Nucleic acid

Since ROS is generated in mitochondria, the susceptibility of oxidative damage to the mitochondrial DNA is higher than nuclear DNA. Nitrogenous bases, as well as deoxyribose sugar, are adversely affected creating single and double-stranded breaks. Several adducts are formed by the attack of OH• radicals on purine and pyrimidine such as 5-hydroxy-6-hydro-cytosine, 8-hydroxydeoxy guanosine, 5-formyl uracil, etc. Of all the free radical-induced adducts, the presence of 8-hydroxydeoxy guanosine indicates oxidative DNA damage [3, 5].

Due to the single-stranded structure, RNA is more adversely affected by oxidative stress than DNA. The most studied adduct of RNA is the 7, 8-dihydro-8-oxo-guanosine (8-oxoG) and its linkage has been established with Alzheimer's, Parkinson's and other neurodegenerative diseases [2, 5].

3.2. Proteins

ROS mediated protein oxidation is usually determined by the presence of carbonyl groups. Different amino acids in the protein might get affected leading to conformational changes which may alter or decrease the function of the oxidized proteins [3, 4].

3.3. Lipids

Membrane lipid peroxidation is one of the conditions which may lead to a series of disorders. The decrease in membrane fluidity is observed during lipid peroxidation which further leads to the inactivation of membrane-bound proteins [4].

4. Effect of oxidative stress on human health

Oxidative stress has been known to promote the induction of several acute and chronic disorders some of which may be degenerative or fatal (Table.1).

4.1. Cancer

Oxidative stress can cause direct damage to various biomolecules. Oxidative DNA damage has now been proven to be a prerequisite in chromosomal abnormalities and oncogene activation thereby causing tumor genesis and/or carcinogenesis [6]. DNA-protein crosslinks, deformity in sugar and base structure are few of the structural changes induced by ROS. According to Hattori et al., 8-hydroxy-2-deoxyguanosine is a suitable biological marker for oxidative stress. Incessant oxidative stress may affect the proteome to cause an alteration in the protein structure [7]. This may generate abnormalities in the structure and function of the proteins such as phosphatases and kinases, Loss, gain or switch in function of these enzymes lead to uncontrolled cell growth [8]. Oxidative stress has been correlated with cancer mainly of the breast, colon and prostate [4].

4.2. Cardiovascular diseases

Oxidative stress acts as a triggering component for the formation of cholesterol plaque in the walls of arteries. This condition is called atherosclerosis and cause an obstruction in the blood flow [9]. Moreover, augmented levels of superoxide anions have been reported to have a direct effect on the pathogenesis of atherosclerosis. One of the theories suggests that these superoxide anions cause oxidative modifications in low-density lipoproteins (LDL) leading to atherosclerotic lesions and lipid accumulation [10].

4.3. Diabetes

High level of sugar in blood or hyperglycemia is known to elevate ROS levels leading to discrepancies in the normal functioning of metabolic pathways. One such example is the decrease in the activity of glyceraldehyde 3-phosphate dehydrogenase (GAPDH) which is one of the key enzymes of

one of the major carbohydrate catabolism, i.e. Glycolysis, by modifying it with ADP-ribose polymers [11]. The islets β-cells of the pancreas get adversely affected when interacted with hydrogen peroxide and superoxide anions that cause lower or inefficient insulin activity [12].

4.4. Inflammation

Activation of transcription factors like NF-kappa B causes inflammation when stimulated by oxidants. Biswas has stated inflammation and oxidative stress to be a tightly linked pathophysiological process wherein one can act as the inducing factor for the other [13].

Endothelial dysfunction and tissue injury can occur at the site of inflammation with an increased number of ROS [14]. This can be exemplified with the pathogenesis of rheumatoid arthritis, the chronic inflammation of joints and the surrounding tissues, which is caused due to the formation of ROS and RNS at the inflammatory site [15].

4.5. Neurodegenerative disorders

The high lipid content, as well as a high level of oxygen consumption by the Central Nervous System (CNS), increases its susceptibility to oxidative stress. The decrease in membrane fluidity by lipid peroxidation increases the permeability of Ca^{2+} which affects the triggering of neurotransmitter release [16]. Huntington's, Alzheimer's, Parkinson's disease, amyotrophic lateral sclerosis (ALS), memory loss, depression, and multiple sclerosis are some of the few diseases resulting from oxidative stress [1, 5].

4.6. Obesity

A decrease in vasodilatory response to acetylcholine in obese patients has been observed as a result of the induction of oxidative stress [17]. Overweight leads to irregularity in the function of adipose tissue, which in turn facilitates hyperglycemia acting as a contributing factor in type-2 Diabetes mellitus [18]. Furukawa et al. propose that the chance of obesity-associated metabolic syndrome is proportional to the increase in the level of oxidative stress [19].

4.7. Respiratory diseases

Inflammation of the respiratory tract has been associated with periodic worsening of asthma**.** It has been observed that there has been an increase in hydrogen peroxide and isoprostanes levels in sputum and exhaled air during an allergic reaction [20].

Table 1. Diseases caused due to Oxidative stress

Disease/ Disorder	Particulars	References
Cancer	Colorectal, breast, prostate	[5, 6]
Cardiovascular diseases	Atherosclerotic lesions and lipid accumulation	[9, 10]
Diabetes	Lower or inefficient insulin activity	[11, 12]
Inflammation	Endothelial dysfunction and tissue injury, Rheumatoid arthritis	[13, 14]
Neurodegenerative disorders	Huntington's, Alzheimer's, Parkinson's disease, ALS, memory loss, depression, multiple sclerosis	[4, 16]
Obesity	Obesity-associated metabolic syndrome	[17, 18]
Respiratory diseases	Asthma	[20]

5. Antioxidants

Antioxidants are the substances that help to maintain the stability in oxidative stress by preventing oxidation [21]. The human body produces a wide range of antioxidants which can be grouped into enzymatic and non-enzymatic antioxidants (Figure 2).

Superoxide dismutase or SODs (EC 1.15.1.11), catalases (EC 1.11.1.6) and glutathione peroxidase or GSH-Px (EC 1.11.1.9) are the major enzymatic antioxidants in the body. SODs are widely expressed in the lungs and help in the dismutation of superoxides. Of the three types of SODs, MnSOD and CuZn-SOD are expressed in mitochondrial and extracellular matrix respectively, while certain SODs are expressed extracellularly and hence called EC-SOD. Catalases and GSH-Px help in the reduction of H_2O_2 produced during the action of SODs or during ETC [21, 22].

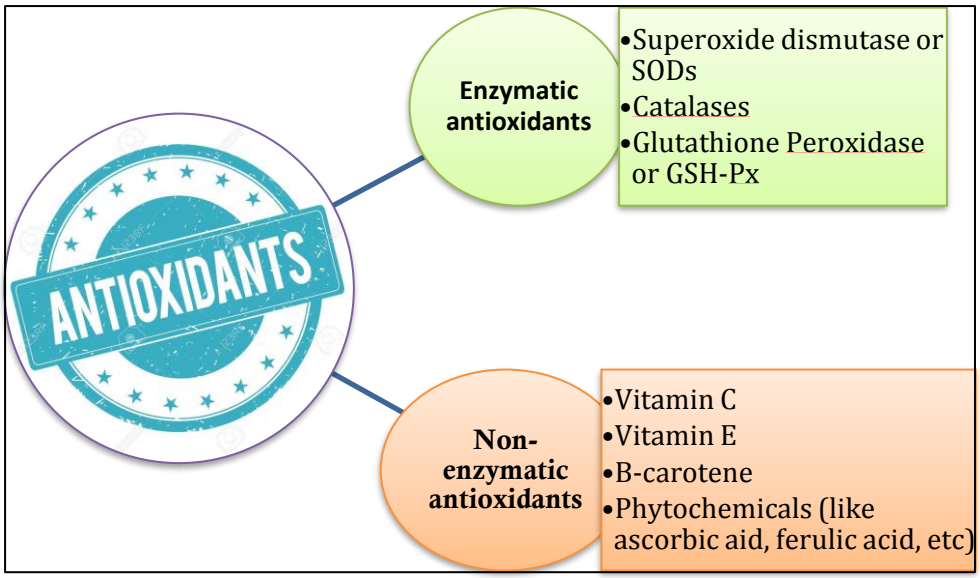

Figure 2. Types of antioxidants

Vitamin C is one of the important non-enzymatic antioxidants in the body. Being water-soluble, it provides an aqueous phase for free radical scavenging. α-Tocopherol OR Vitamin E is a membrane-bound active form of vitamin E antioxidant which inhibits free radical formation. Phytochemicals in the plant have been proven to show antioxidant activity. B-carotene acts as an effective antioxidant against peroxyl (ROO•), hydroxyl (•OH), and superoxide (O_2^{-}) radicals [22, 23]. The phytochemicals are being tested for suitability as it appears to be a promising candidate for antioxidant supplements [23].

6. Conclusion

Over-production of oxidants or unavailability of antioxidants can cause an imbalance in the free radical generation and their detoxification leading to oxidative stress. Oxidative stress seems to be playing a major role in inflammation that further acts as a contributing factor to a plethora of diseases. The human body can combat oxidative stress by maximizing the availability of natural antioxidants. However, antioxidants can be supplemented to the human body from a wide range of drugs synthesized from chemical as well as natural sources. However, being environmentally friendly, natural antioxidants like phytochemicals are considered to be more convenient and safer for consumption.

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