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Protective Role of Some Vitamins Against DNA Damage: An Electrochemical Approach

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Bazı Vitaminlerin DNA Hasarına Karşı Koruyucu Rolü: Elektrokimyasal Bir Yaklaşım

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Abstract

This study investigates the electrochemical detection of DNA damage induced by infrared (IR) radiation and evaluates the protective effects of vitamins C, D, E, and B7 (biotin) against such damage. DNA was immobilized onto a pencil graphite electrode (PGE), and variations in the guanine oxidation signal were monitored by differential pulse voltammetry (DPV) in 0.5 M acetate buffer (pH 4.8) using a three-electrode system (PGE/Ag–AgCl/Pt). DNA-modified electrodes (DNA–PGE) and vitamin-coated DNA–PGEs, prepared by incubating the DNA–PGEs in vitamin solutions, were exposed to IR radiation for 1 h. In the absence of vitamins, IR exposure caused a marked decrease in the guanine oxidation peak current from 1.55 µA to 0.67 µA (a 57% reduction), indicating substantial DNA damage. In contrast, electrodes coated with vitamins D and B7 exhibited statistically significant protection ($p < 0.05$), as evidenced by a significantly reduced decrease in the guanine oxidation signal following IR exposure. Vitamins C and E, however, did not show a significant protective effect. These findings suggest that vitamins D and B7 may serve as potential protective agents against IR-induced genotoxic damage.

Keywords: *DNA damage, Electrochemistry, Guanine, Vitamin, IR rays.*

Öz

Bu çalışmada, kızılötesi (IR) ışınlarının neden olduğu DNA hasarının elektrokimyasal olarak belirlenmesi ve C, D, E ile B7 (biotin) vitaminlerinin bu hasara karşı koruyucu etkilerinin araştırılmasını amaçlamaktadır. DNA, kalem grafit elektrot (PGE) yüzeyine immobilize edilmiş ve vitamin varlığında ve yokluğunda guanin yükseltgenme sinyalindeki değişim 0.5 M asetat tamponunda (pH 4.8) üçlü elektrot sistemi (PGE/Ag–AgCl/Pt) kullanılarak diferansiyel puls voltametri (DPV) tekniği ile izlenmiştir. DNA immobilize edilmiş elektrotlar (DNA-PGE) ve üzeri vitamin çözeltileri içinde bekletilerek vitamin ile kaplanmış DNA-PGE'ler 1 saat süre ile IR ışınlarına maruz bırakılmış ve sonuç Guanin yükseltgenme sinyalindeki değişimler izlenerek takip edilmiştir. Vitamin yokluğunda IR ışınlarına 1 saat maruziyet sonunda guanin yükseltgenme sinyali 1.55 µA'dan 0.67 µA'a düşmüştür (%57 azalma). Vitamin D ve B7 ile kaplanmış DNA-PGE'ler ile IR maruziyeti sonrasında ölçülen guanin yükseltgenme sinyali, bu vitminlerin IR ışınına karşı koruyucu davrandığını ve guanin sinyalindeki azalmayı istatistiksel olarak anlamlı biçimde önlediğini ($p < 0.05$) göstermiştir. Buna karşın vitamin C ve E ile kaplanmış DNA-PGE'lerin 1 saatlik IR maruziyeti sonrasında ölçülen guanin yükseltgenme sinyalinde belirgin bir koruyucu etki gözlenmemiştir. Bulgular, D ve B7 vitaminlerinin IR kaynaklı genetik hasara karşı potansiyel koruyucu ajanlar olarak değerlendirilebileceğini göstermektedir.

Anahtar Kelimeler:*DNA hasar, Elektrokimya, Guanin, Vitamin, IR ışınları.*

1. Introduction

Deoxyribonucleic acid (DNA) has been recognized as a central molecular target for various forms of damage that can disrupt cellular functions, leading to the development of serious health conditions, including cancer and degenerative diseases. Environmental factors such as oxidative stress, environmental toxins, infrared ray (IR), and replication errors pose a significant threat to the structural integrity of DNA, often resulting in mutations, chromosomal abnormalities, and genetic instability (Lam 2022, Hanahan and Weinberg 2011). This disruption can trigger tumorigenesis, contributing to cancer and other diseases characterized by genomic instability (Chapman et al. 2012, Lieber 2010).

Among the various types of DNA damage, double-strand breaks (DSBs) are considered to be one of the most critical and damaging forms. DSBs are highly cytotoxic and can result in cell death if left unrepaired, making them a severe threat to genomic integrity (Shiloh and Ziv 2013). To counteract these risks, cells have evolved intricate DNA repair mechanisms that involve signaling pathways activated by key proteins such as ataxia-telangiectasia mutated (ATM) and Rad3-related (ATR) kinases, which help coordinate the repair process (Martejin et al. 2014).

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Furthermore, DNA repair can be influenced by a range of cellular factors, including epigenetic modifications, chromatin remodeling, and metabolic changes (Talib et al. 2024). Despite extensive research in this area, significant gaps remain in our understanding of how DNA damage can be prevented and how repair mechanisms can be enhanced.

Cancer is closely linked to DNA damage, and it remains one of the leading global health challenges. Over the past decades, considerable research has been dedicated to identifying strategies for both the prevention and treatment of cancer. Of particular interest in recent years has been the potential role of vitamins in modulating DNA damage and repair mechanisms. Certain vitamins, including vitamin D, vitamin C, vitamin E, and vitamin B_7, have been implicated in modulating key biological processes involved in cellular stress response, DNA repair, and carcinogenesis (Jain et al. 2017).

Vitamin D may play a protective role against DNA damage. Studies have shown that vitamin D supplementation can reduce levels of 8-hydroxy-2'-deoxyguanosine (8-OHdG), a marker of oxidative DNA damage, in colorectal epithelial cells. Animal models and cell culture studies also indicate that vitamin D decreases oxidative stress–induced DNA damage, chromosomal abnormalities, and telomere shortening, while inhibiting telomerase activity. The protective effect of vitamin D against DNA damage appears to be primarily through prevention of oxidative stress and secondarily via regulation of the cell cycle and induction of apoptosis. However, there is limited data on the optimal dose and duration of vitamin D supplementation needed to minimize DNA damage in humans (Nair-Shalliker et al. 2012).

Vitamin D exhibits antitumorigenic properties. In cell and animal models, it has effects on suppressing tumor growth, promoting cell differentiation, increasing apoptosis, and reducing inflammation (Jeon and Shin 2018).

Numerous epidemiological studies have established a link between higher vitamin D levels and a reduced risk of several cancers, including colorectal, breast, prostate, and pancreatic cancers (Keum and Giovannucci 2014, Grant2011). Mechanistically, vitamin D exerts its anticancer effects through various pathways, including cell cycle regulation, inhibition of angiogenesis, and promotion of apoptosis (Powala et al. 2024).

Similarly, vitamins C and E, known for their potent antioxidant properties, have been proposed to exert protective effects against DNA damage by neutralizing reactive oxygen species (ROS) and free radicals. These antioxidants are thought to mitigate the damage caused by oxidative stress, which is a well-established risk factor in the development of various cancers. Several studies have demonstrated the ability of vitamins C and E to lower the risk of cancers such as gastric, breast, lung, and colorectal cancers (Villagran et al. 2021, Myung et al. 2010, Gaziano et al. 2009). Additionally, recent investigations have explored the potential of high-dose supplementation with these vitamins as an adjuvant to traditional cancer therapies, such as chemotherapy and radiation therapy. Notably, these studies have shown promising results, including enhanced tumor response and improved survival outcomes in some patient cohorts (Gaziano et al. 2009, de Oliveria et al. 2009).

Vitamin B7 (biotin) has been shown to reduce DNA strand breaks and protect genomic integrity under stress conditions. Its protective effect is attributed, in part, to its antioxidant properties, which mitigate reactive oxygen species (ROS)-induced oxidative damage and enhance cellular defense mechanisms (Ranjan et al. 2022). Emerging research suggests that vitamin B7 transporters are highly expressed in certain cancer cells, implying a possible link between vitamin B7 metabolism and cancer progression (Zempleni et. al. 2012). Furthermore, innovative therapeutic approaches incorporating vitamin B7, such as vitamin B7-conjugated drug delivery systems, are being explored for targeted cancer treatment strategies (Wang et al. 2025). These findings point to the expanding role of vitamin B7 in cancer research and its potential therapeutic applications.

Among the various cancers, skin cancer is one of the most prevalent types worldwide, and it is directly linked to DNA damage, especially damage induced by infrared ray (IR). The connection between IR exposure and skin cancer underscores the importance of developing effective, non-invasive methods for detecting DNA damage, particularly in relation to skin cancer. Electrochemical methods have emerged as a reliable and sensitive approach for detecting DNA damage, offering a promising alternative to traditional diagnostic techniques. These methods enable real-time monitoring of DNA damage and have been explored for their potential in early cancer detection and monitoring therapeutic responses.

In the context of IR-induced DNA damage, this study aimed to investigate the protective effects of various vitamins using electrochemical biosensor technology. A biosensor was developed by immobilizing DNA onto a pencil graphite electrode surface, allowing for the electroanalytical detection of DNA damage and the

evaluation of the potential protective effects of vitamins. Through these innovative approaches, the study provides valuable insights into the mechanisms of DNA damage, the role of vitamins in DNA repair, and the potential for developing novel strategies for cancer prevention and treatment. The results contribute to the growing body of evidence on how vitamins can modulate DNA damage and repair and open new avenues for research into therapeutic strategies targeting DNA damage-related diseases.

Recent studies have further illuminated the role of vitamins in DNA damage prevention and repair, highlighting their potential as protective agents against cancer and other diseases associated with genomic instability. The integration of electrochemical techniques in this research provides a promising platform for future investigations into the dynamic interaction between vitamins and DNA repair mechanisms, offering new possibilities for enhancing cancer therapies and improving patient outcomes.

2. Materials and Methods

2.1. Apparatus

Electrochemical measurements were conducted using the IVIUM Compact Stat analyzer (Netherlands) in a three-electrode system. In this system, a pencil graphite electrode (PGE) (Tombow, type HB, 0.5 mm diameter, Live Co., Japan) served as the working electrode, an Ag/AgCl/KCl (3 M) electrode (BAS, Model RE-5B, W. Lafayette, USA) was used as the reference electrode, and a platinum wire functioned as the auxiliary electrode.

2.2. Chemicals

Vitamins D and E were obtained from a pharmacy, while all other chemicals were purchased from Sigma-Aldrich. The chemicals used were of HPLC or analytical reagent grade and were used without further purification. Ultrapure water, obtained from a Millipore purification system, was used for the preparation of all solutions.

Fish sperm DNA (fsDNA) was used in this study as the double-stranded DNA. Stock DNA solution was prepared in Tris(hydroxymethyl)aminomethane (Tris)-Ethylenediaminetetraacetic Acid (EDTA) buffer (10 mM Tris-HCl, 1 mM EDTA, pH 8.00) at a concentration of 1000 µg/mL and stored at - 20°C. All required dilutions were prepared using an acetate buffer (ABS, pH 4.8), which was obtained by adjusting 0.5 M acetic acid with sodium hydroxide.

Vitamins D and E were investigated at their commercially available concentrations. Stock solutions of vitamin C and vitamin B7 were freshly prepared each day by dissolving the compounds in an ethanol-water (C_2H_5OH-H_2O) mixture (1:1) at a concentration of 10 mM and 200 ppm respectively followed by sonication for 15 minutes.

2.3. Procedure

2.3.1. Activation of pencil graphite electrodes and electrochemical measurements

The pencil graphite electrodes (PGEs) used in this study were fabricated by cutting the pencil tips to a length of 3.0 cm. Prior to analysis, the electrode surfaces were electrochemically activated by applying a potential of +1.4 V for 30 s in an acetate buffer solution (ABS).

All voltametric measurements were performed by differential pulse voltammetry (DPV) technique in 0.5 M phosphate buffer solution (PBS, pH: 7.0) by scanning in the potential range from 0 to+1.5 V at the 50 mV pulse amplitude and 50 mV/s scan rate.

2.3.2. Damage studies

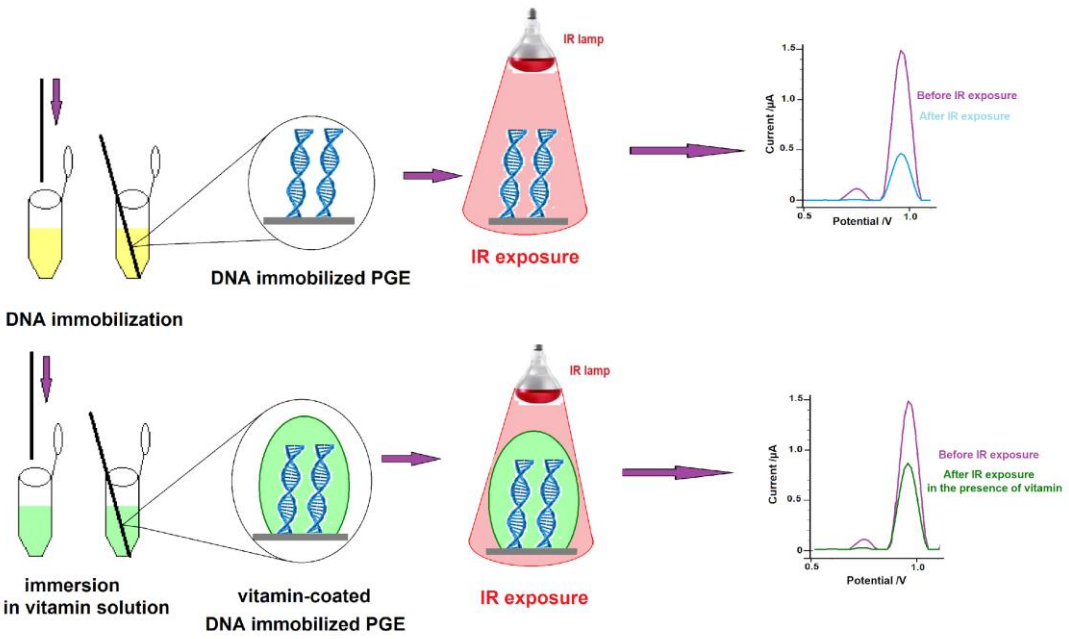
DNA was immobilized onto the surfaces of two distinct groups of electrodes. For the first group, electrochemical measurements were carried out directly by anodic scanning. In contrast, the second group of electrodes was subjected to IR lamp exposure for 1 h prior to anodic scanning. Guanine was chosen as the analytical target due to its lower oxidation potential and superior sensitivity relative to the other nucleobases.

In experiments conducted in the presence of vitamins (B7, C, D, and E), electrodes with immobilized DNA were immersed in vitamin solutions at specific concentrations for 1 hour, followed by exposure to an IR lamp for an additional hour. Subsequently, guanine oxidation signals were measured using the DPV technique. Experimental procedure was also illustrated in Figure 1.

3. Results and Discussion

In the DNA damage study involving IR exposure, the concentration of immobilized DNA on the electrode surface must be maximized, as the damage is induced by subjecting the electrodes to an IR lamp. Based on our previous studies, a DNA concentration of 80 µg/mL was applied to ensure full coverage of the electrode surface (Öndeş and Muti 2020, Erol et al. 2021, Çalıcıoğlu et al. 2025).

To examine the effect of IR irradiation, different exposure times were applied to the 80 µg/mL fsDNA immobilized electrodes. The percentage decrease in the guanine oxidation signal after 1 hour of IR exposure is illustrated in Fig. 1.



Scheme 1. Schematic presentation of the IR exposure and the electrochemical measurement process.

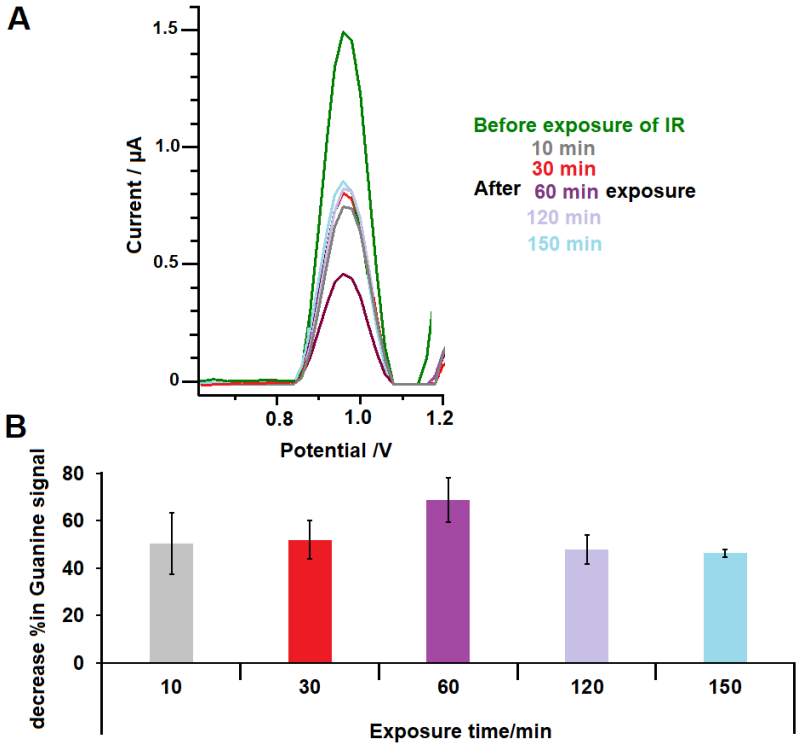


Figure 1. (A) Voltamograms showing the guanine oxidation signal before and after IR exposure for 10, 30, 60, 120, 150 min. (B) Bar graphs illustrating the percentage decrease in the guanine oxidation signal with increasing IR exposure duration.

The most significant decrease in the DNA signal was observed after 1 hour of exposure (Before exposure: 1.55 µA, RSD % = 2.3, n = 8; After 1 hour of exposure: 0.65 µA, RSD % = 9.3, n = 8). Based on these findings, it was determined that future studies should incorporate 1 hour of IR exposure.

Four groups of electrodes were prepared, with 80 µg/mL of fsDNA immobilized on their surfaces. The first group of electrodes was directly measured, the second group of electrodes was exposed to an IR lamp for 1 hour before measurement. The third group was immersed in a vitamin solution for 1 hour, followed by measurement. In the fourth group, the electrodes were first immersed in the vitamin solution and then kept under IR lamp for 1 hour prior to electrochemical measurements. Vitamins B_7, C, D and E were used in the damage studies to investigate their role in preventing DNA damage. The results obtained are shown in Figure 2, 3, 4 and 5 respectively.

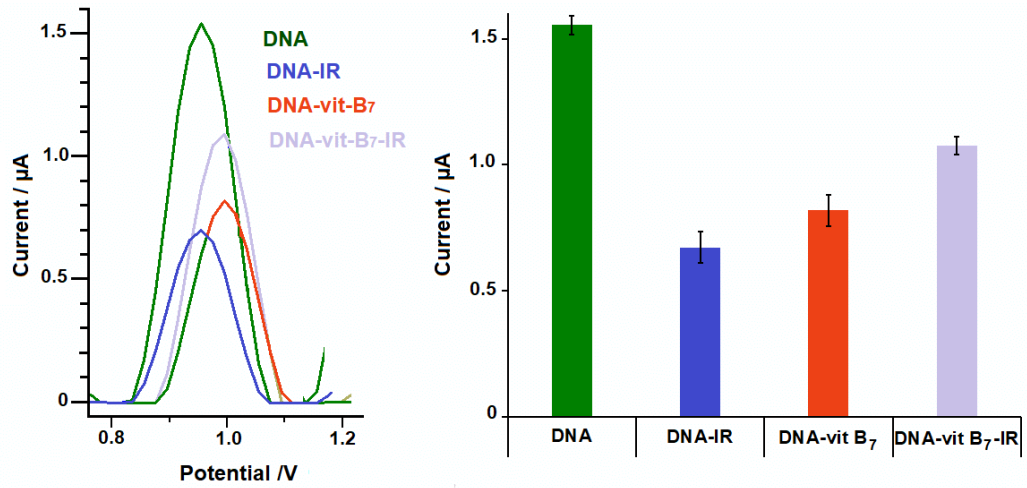


Figure 2. Voltammograms illustrating the guanine oxidation signals after 1 hour of IR irradiation of 80 µg/mL fsDNA-immobilized electrodes in the absence and presence of vitamin B_7 (0.2 mg/mL) along with a histogram generated by averaging the signals.

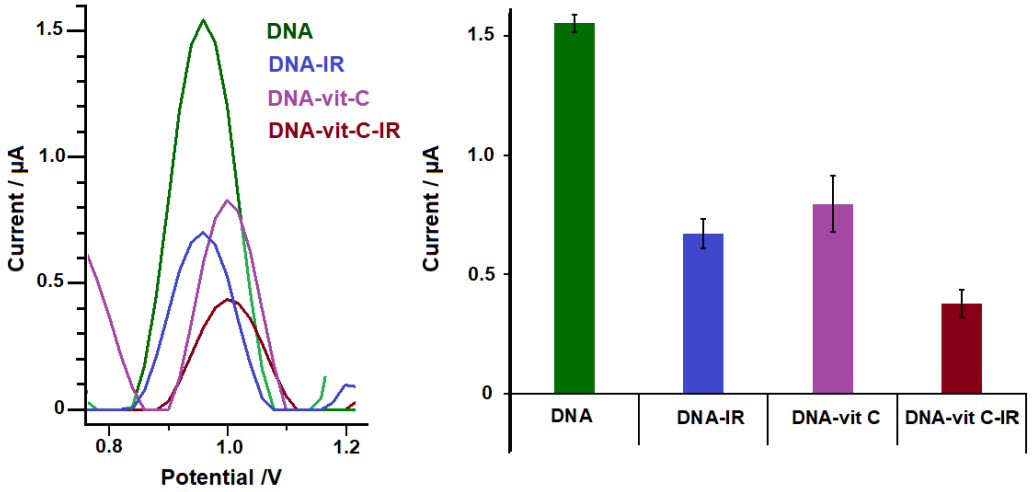


Figure 3. Voltammograms illustrating the guanine oxidation signals after 1 hour of IR irradiation of 80 µg/mL fsDNA-immobilized electrodes in the absence and presence of vitamin C (10 mM) along with a histogram generated by averaging the signals.

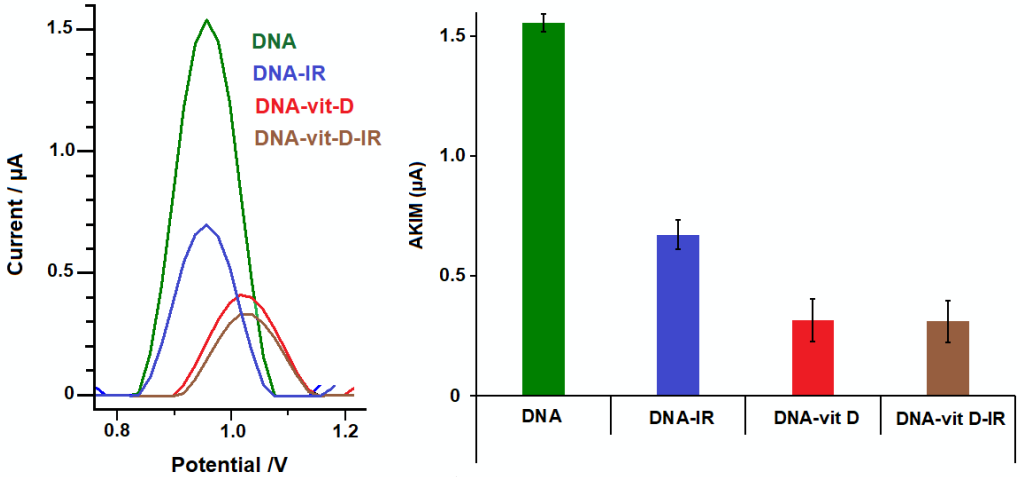


Figure 4. Voltammograms illustrating the guanine oxidation signals after 1 hour of IR irradiation of 80 µg/mL fsDNA-immobilized electrodes in the absence and presence of vitamin D (30,000 I.U/mL) along with a histogram generated by averaging the signals.

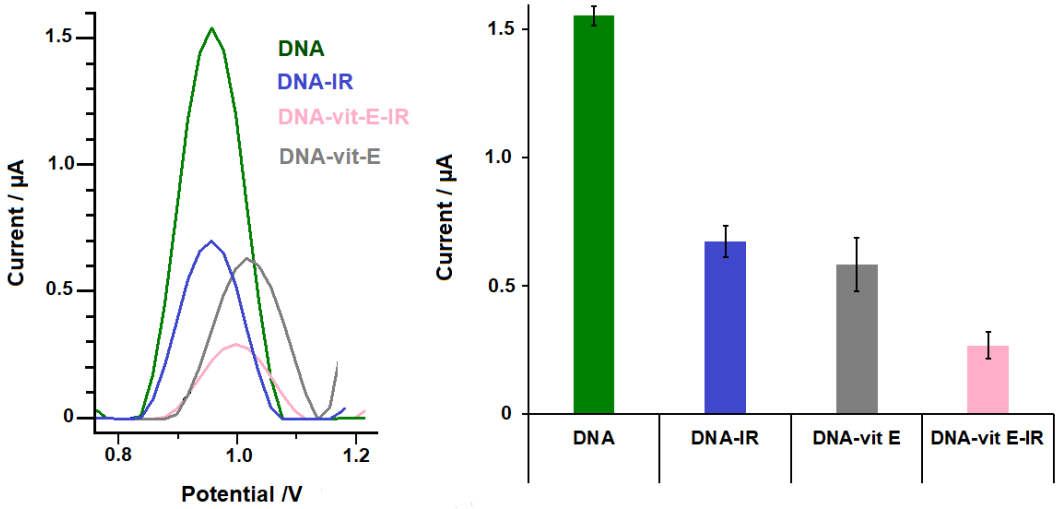


Figure 5. Voltammograms illustrating the guanine oxidation signals after 1 hour of IR irradiation of 80 µg/mL fsDNA-immobilized electrodes in the absence and presence of vitamin E (150 mg/mL) along with a histogram generated by averaging the signals.

The results obtained in the absence and presence of vitamins were also illustrated in Table 1.

Table 1. Guanine oxidation signals after 1 hour of IR irradiation of 80 µg/mL fsDNA-immobilized electrodes, both in the absence and presence of vitamins

	Signal (µA)	RSD%
DNA	**1.55**	2.5, (n=8)
DNA-IR	0.67	9.3, (n=8)
DNA-vit C	0.80	14.8, (n=3)
DNA-vit C-IR	0.38	15.2, (n=3)
DNA-vit D	0.32	28.3, (n=3)
DNA-vit D-IR	0.31	28,3, (n=3)
DNA-vit E	0.58	18.0 (n=3)
DNA-vit E-IR	0.27	20.0, (n=4)
DNA-vit B_7	0.82	7.6 (n=4)
DNA-vit B_7-IR	1.08	3.3 (n=3)

The values in the table indicate that vitamins C and E do not have any effect on preventing IR damage, while vitamins D and B7 are effective in preventing IR damage.

The anodic shift observed in the guanine oxidation signal after coating the DNA-immobilized electrode surface with vitamins (B_7, C, D, E) is likely due to the formation of a barrier layer that hinders electron transfer between guanine residues and the electrode, while electrostatic interactions introduced by the vitamin layer may further increase the oxidation potential, resulting in the positive shift.

4. Conclusion

Approximately 55% of solar radiation consists of infrared (IR) rays. For individuals who must work under direct sunlight or reside in regions with intense solar exposure, identifying and preventing the adverse effects of IR radiation on the skin is of critical importance.

The findings of this study demonstrate that vitamins may exert protective effects against DNA damage through diverse mechanisms. Vitamin D has previously been reported to play a regulatory role in cellular stress responses and DNA repair pathways. In this study, its contribution to maintaining DNA integrity has been experimentally confirmed. Similarly, vitamin B7 provides compelling evidence for a role beyond its well-established metabolic functions, indicating a capacity to prevent IR-induced DNA damage. The use of electrochemical biosensors enables rapid, sensitive, and reproducible detection of DNA damage, thereby highlighting potential applications in future diagnostic and screening technologies. Future research should focus on exploring the synergistic effects of different vitamin combinations and elucidating the underlying molecular mechanisms of their protective actions. Such efforts may contribute to the development of skin-applied cosmetic products aimed at mitigating the harmful effects of IR radiation.

In conclusion, this study provides significant evidence that IR-induced DNA damage can be alleviated through vitamin intervention, underscoring the potential of vitamin D and biotin as promising candidates in DNA protection mechanisms.

Declaration of Ethical Standards

The authors declare that they comply with all ethical standards.
This study is derived from master thesis (thesis number: 746878) under the supervision of Prof. Dr. Mihrican Muti by Pelin Uysal Çalıcıoğlu on date of 29/06/2022. Title: " Determinaation of DNA damage caused by IR rays by electrochemical methods and investigation of the damage in the presence of some molecules"

Credit Authorship Contribution Statement

Author-1: Conceptualization, investigation, methodology and software, supervision and writing – review and editing.
Author-2: Writing—original draft, investigation, visualization.
Author-3: Investigation, software, formal analysis.
Author-4: Conceptualization, investigation, methodology, supervision.

Declaration of Competing Interest

The authors have no conflicts of interest to declare regarding the content of this article.

Data Availability Statement

All data generated or analyzed during this study are included in this published article.
Datasets are available on request.

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