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The Role of Sunscreen in Mitigating UV-Induced Damage in *Saccharomyces cerevisiae* Yeast Respiration and Survival, as a Eukaryotic Model Organism

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Abstract

Mutagens are agents that cause damage to DNA and have the potential to permanently alter (mutate) its sequence, depending on the organism's ability to repair the damage. UV radiation is a mutagen in cells. This mutagen relates to both yeast cells and human skin cells, since they have similar reactions. UV radiation can cause cell mutations, but also cell death. This is examined with the absence or presence of sunscreen when in contact with cells. Since yeast cells and human cells have almost identical metabolisms, data results of yeast experiments can be associated with real life. Hypothesis for the presence of sunscreen in yeast solutions includes the exposure of yeast cells with or without sunscreen for different time periods in UV radiation. However, the role of sunscreen in yeast cell mutations, in relation to cancer prevention, may not be directly positive. Here, I show that sunscreen has a positive effect on yeast cells and prevents mutations. I found that the respiration rate differs for yeast cells without or without the presence of sunscreen when exposed to radiation. Yeast cells without sunscreen respired faster than those exposed to UV radiation. However, with sunscreen, the rate of CO_2 production was higher, with a higher respiration rate. These results may be connected with skin cancer to some extent, promoting or not the use of sunscreen to protect the skin cells from mutating. This experiment may be the base for further experimentation with different yeast cells, providing clearer and more assuring data about the association of sunscreen, yeast cells, and skin cancer. Such experiments may avoid implications with weather conditions, such as slightly different temperatures, sunlight intensity, and clouds, or with the time between the end of the time period of exposure of the yeast cells to UV radiation, and the measurement of CO_2 and density, which my experiment had.

Keywords

UV Radiation, Yeasts (Yeast Cells), Skin Cells, Cell Respiration (CO_2 Rate, Cell Density)

1. Introduction

1.1. Yeasts

Yeasts, a group of around 1500 single-celled fungi species, are found in sugary media like fruit nectar and flower nectar, and have been traditionally used in the production of bread, beer, and wine [1]. They are eukaryotic organisms with a diameter of around 0.075 mm and can be spherical, egg-shaped, or filamentous [1]. Most yeasts reproduce asexually through budding, while some split into two equal cells through fission [1].

Yeasts are used in food production, where they produce carbon dioxide and ethanol through fermentation. These byproducts are used in bakery products, beer, and wine making. Yeast cells can ferment about their own weight of glucose in an hour [1]. Yeast for baking is available in two forms: compacted cakes with starch or dry grains mixed with cornmeal [1]. Commercial yeast is rich in niacin, folic acid, vitamins B1, B2, and B2, and has a 50% protein content. Deactivated brewer's yeast and nutritional yeast can be taken as vitamin supplements [1].

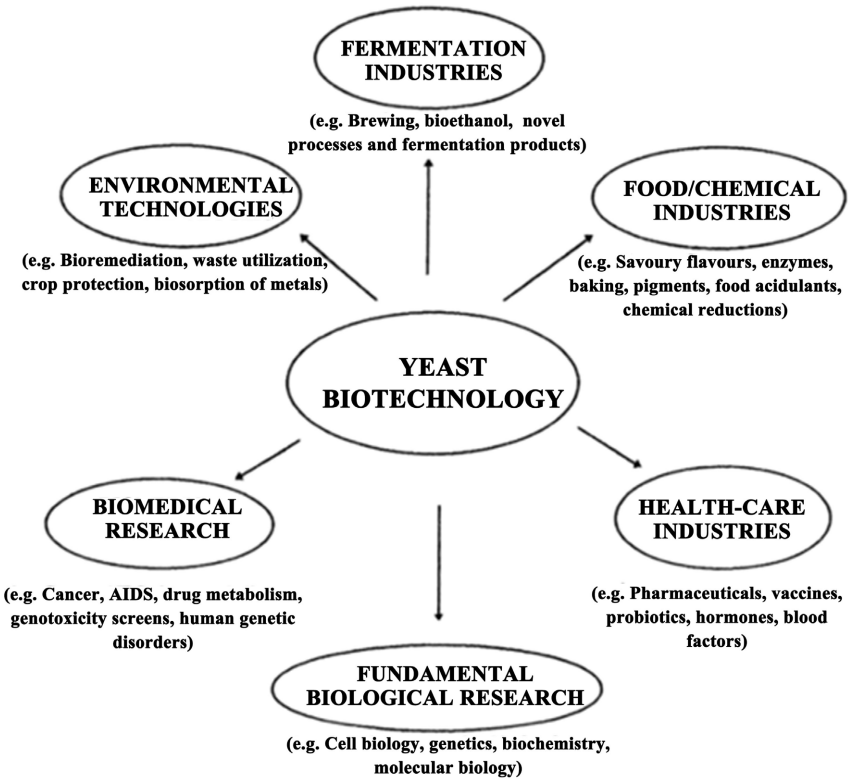

Figure 1. Diversity of outlets involving yeast biotechnology roles [3].

Yeast identification involves physiological and morphological assays, including auxanography for determining carbon and nitrogen sources. Auxanography is a study that determines the growth of mutants which require specific substances to develop [2]. Systems like BCCM/Allev 2.00 and API strips analyze sugar absorption and fermentation. Yeasts inhabit diverse environments such as plant tissues, air, water, and land, with some thriving in solute-rich, extreme conditions. Species like *S. cerevisiae* colonize wine, while others are found in hospitals or cause food spoilage [3] (**Figure 1**).

Saccharomyces cerevisiae relies on fermentation for energy, even in the presence of oxygen. When glucose is scarce, it switches to using ethanol as a carbon source, triggering a shift in gene expression to favor gluconeogenesis (metabolic reactions that maintain blood glucose levels constant after digestion) [4] and the glyoxylate cycle (a variant of the tricarboxylic cycle found in plants, fungi and protists, permitting the use of two carbon compounds, when glucose is not present) [5], while reducing fermentation-related genes. Zinc cluster proteins like Cat8, Sip4, Rds2, and Adr1 drive this gene reprogramming.

$NAD^+ + 2e^- + H^+ \rightarrow$ Reduced NAD ($NADH + H^+$) (**Figure 2**)

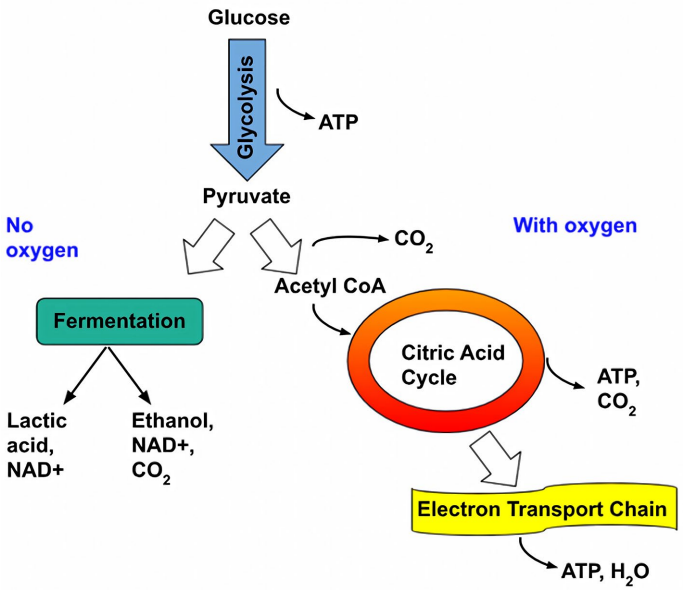

Figure 2. Cellular respiration and fermentation overview [6].

1.2. Sunscreens

The standard erythema dose (SED) measures the biological efficiency of UV radiation, specifically its ability to cause erythema (skin reddening). Sunscreens, although often applied less thoroughly than in SPF testing, can reduce sunburn cells, DNA damage, and risks of skin cancer such as actinic keratoses and squamous cell carcinomas. Chronic UV exposure is the leading cause of malignant melanoma and contributes to both photoaging and photo carcinogenesis [7]. Inorganic sunscreens like zinc oxide and titanium dioxide are effective and increasingly popular

due to improved formulations [7]. SPF remains a key measure of sunscreen efficacy, and recent advancements have combined UV filters with DNA repair agents to enhance skin protection [7].

Extraterrestrial sunlight at sea level includes electromagnetic radiation from 290 to 3000 nm, with UV radiation categorized into UVA (320 - 400 nm), UVB (290 - 320 nm), and UVC (200 - 290 nm). While UVA predominates at the Earth's surface, UVC is fully absorbed by the ozone layer. Variations in UV radiation depend on latitude, time of day, and season. The depletion of the ozone layer due to pollutants like nitric oxides and chlorofluorocarbons could increase UVB and UVC exposure, raising risks of skin cancer, photo immunosuppression, premature aging, and photosensitive diseases [8].

UV radiation, prevalent in the environment, contributes to skin diseases like inflammation, aging, and cancer. Personal exposure depends on sunlight intensity, time outdoors, and protective measures like clothing and sunscreen [9].

UVB and UVA radiation damage skin biomolecules, with UVB directly causing DNA lesions that can lead to mutations and skin cancer, particularly involving p53 mutations. The majority of p53 mutations are missense mutations, which produce full-length mutant p53 proteins. In addition to losing their ability to inhibit malignancies in a way dependent on wild-type p53, mutant p53 (Mutp53) proteins frequently acquire oncogenic gain-of-functions (GOF) that promote tumor growth [10]. UVA contributes to photoaging and suppresses the immune system. Photoprotection, including sun avoidance, protective clothing, and sunscreen, is central for preventing skin damage, photo immunosuppression, and skin cancers, and has become a major public health approach [10] [11].

Octinoxate, a common UVB absorber, is well-tolerated but degrades under sunlight, reducing its effectiveness. Encapsulation in nanoparticles can enhance their photostability. Avobenzone (Parsol 1789) is a strong UVA filter, but stabilizers may be needed to prevent degradation. Benzophenone-3, widely used and highly bioavailable, has a higher incidence of photodermatitis. Diethylamino hydroxybenzyl hexyl benzoate, more photostable than avobenzone, offers similar protection [12].

Photostability and Water Resistance

Photostability is essential for sunscreen effectiveness, ensuring it maintains its protective properties under sunlight. Some chemical filters, like octyl dimethyl PABA and avobenzone, can be photoreactive, reducing stability. Other filters, such as zinc oxide (ZnO), titanium dioxide (TiO_2), salicylates, and methyl benzylidene camphor, enhance photostability, helping sunscreens better absorb, reflect, and scatter UV rays while remaining stable [13].

Sunscreen effectiveness in water is assessed by its ability to maintain SPF after immersion. In Europe, sunscreens are classified as "water-resistant" or "extra water-resistant" if post-immersion SPF remains at least 50% of the initial value after 40 or 80 minutes in water. The SPF label in the US reflects the pre-water exposure value [14].

Sunscreens and Melanoma

The link between sunscreen use and melanoma risk is debated, with studies showing conflicting results. Some suggest lower melanoma incidence with sunscreen use [15], while others indicate no significant impact [16]. A meta-analysis found little correlation, likely due to varying study methods and early sunscreens providing only UVB protection [17]. Modern sunscreens with broad-spectrum protection are still essential for preventing sunburn and mutations linked to melanoma, though their effectiveness against melanoma needs further study. Recent trials hint that sunscreens might reduce the risk of developing melanocytic naevi, a melanoma precursor [15] [16].

Cutaneous Responses to UV

UV radiation impacts skin physiology both immediately and over time [18]. Acute effects include inflammation, or "sunburn," triggered by cytokines, which are small proteins that control the development and operation of blood cells and other immune system components [19], and other mediators, leading to keratinocyte apoptosis [19]. UV exposure also causes hyperkeratosis (thickening of the epidermis) and activates damage responses, such as p53-mediated cell cycle arrest and DNA repair [18]. The organism used in this investigation, yeast, has a similar mechanism. Tanning, an adaptive response, increases melanin production to protect against further UV damage, though defects in this process can raise cancer risk. UV light also affects immune function and converts 7-dehydrocholesterol into vitamin D3 [9]. UVA mainly causes oxidative damage, while UVB directly damages DNA, with ongoing research into their effects on the skin [9].

Oxidative Injury

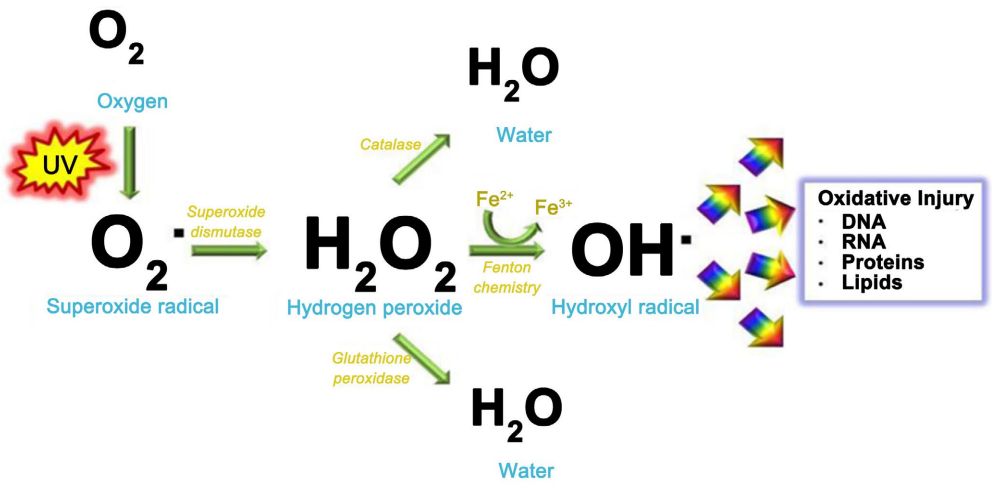

Figure 3. UV photons generate oxidative free radicals, causing structural and functional changes in macromolecules like DNA, RNA, protein, and lipids. Enzymes like glutathione peroxidase, catalase, and superoxide dismutase detoxify these species. Adapted from: https://www.researchgate.net/figure/UV-generates-oxidative-free-radicals-UV-photons-interact-with-atomic-oxygen-to-promote_fig5_237095045

UV light generates reactive oxygen species (ROS), leading to mutations [20].

ROS causes nucleotide damage, resulting in mispairing and mutagenesis, such as the guanine-to-thymine mutation via 8-hydroxy-2'-deoxyguanine (8-OHdG), linked to skin cancer [21]. The base excision repair (BER) pathway repairs DNA damage, with glycosylases identifying and removing altered bases [20]. Antioxidant systems, including glutathione, superoxide dismutase (SODs), and catalase, detoxify ROS, protecting DNA and other macromolecules from UV-induced damage. These processes are crucial in managing the skin's response to UV radiation [20] (**Figure 3**).

2. Research Question

How does exposure time to ambient ultraviolet (UV) radiation (0, 15, 30, 60 minutes) affect the rate of respiration, indicating the death toll of the yeast cells, and survival of yeast (*Saccharomyces cerevisiae*) by measuring carbon dioxide production (in ppm, ±10) and cell viability through the density in spectrophotometer, in the presence and absence of sunscreen?

3. Hypothesis

It is expected that without the presence of sunscreen, as exposure to ultraviolet radiation increases the rate of respiration of yeast and the number of yeast cells surviving will decrease. This is because exposure to UV radiation, damages the yeast cells. This is expected to be more evident at the high exposure time (60 minutes). On the other hand, in the presence of sunscreen it is expected that most yeast cells will survive, and the rate of respiration will also increase. Hence, there will be an increase in the amount of carbon dioxide produced.

4. Variables

4.1. Independent Variables

1) Presence of 9 ml/50ml yeast solution, or absence of Frezyderm Seaside sunscreen UV protection level 50+. The reason that that 9 ml of sunscreen were used per 50 ml of the yeast solution is that it was the minimum amount of sunscreen that created a thin layer on the surface of the solution. The aim was for this surface to be as thin as possible for it to not be embedded in the yeast solution and affect it even more. Also, after having tested different amounts of sunscreen and mixing the sunscreen with the yeast solution, the best outcome was with 9 ml, where only a thin layer was formed. In the other cases, there needed more titrations, leading to a greater error.

2) Time in minutes (0.0 min, 15.0 min, 30.0 min, 60.0 min, ±0.1 min) of exposure to ultra-violet radiation. The solution was placed in natural UV light, so that it could be exposed in the variety of wavelengths and natural conditions, and not only in the UV chamber which has only UVC. Also, these specific time periods were chosen for various of reasons. Firstly, 0.0 was chosen, to have a control variable which would not be exposed to the UV light at all, so that it could be comparable with the rest of the results. Also, this way, there is a better understanding

of if the increase or decrease in CO_2, or in the number of cells, was caused by contact inhibition of the yeast, mutations, or actually due to the UV radiation. More specifically, in contact inhibition, there is the factor of the toxic products that are attained and finally create a toxic environment which causes the death of the cells. Overpopulation is the main cause of this, when mutations may also be fatal.

In Table A1 (**Appendix A**), the ingredients of the sunscreen Frezyderm Seaside sunscreen UV protection level 50+ [22] are presented. To further understand its role, the purposes and the specific wavelength of UV radiation they present are listed.

4.2. Dependent Variables

1) Rate of respiration of yeast (*Saccharomyces cerevisiae*) by measuring the volume of carbon dioxide gas produced (ppm) using a carbon dioxide sensor (±10 ppm). CO_2 will be measured so as to understand if the UV radiation causes cells to die or increases their rate of division. If cells die, then CO_2 will decrease, since the rate of respiration will decrease, and even stop for some cells (the ones that will die). If UV radiation causes mutations, then CO_2 will increase, since more cells will be respiring.

2) Number of yeast cells surviving measured using a spectrophotometer (absorbance). This number will be measured to understand if the UV radiation causes cells to die or mutate and increase in number. To convert absorbance to density, $N = 0.125 * r$ was used, where N is the density, and r is the number of times which the sample is diluted [23]. If UV radiation kills the cells, then the density after exposure will decreases. On the other hand, if it causes mutations, then the cell density will increase.

In Table 1, the controlled variables are stated. Specifically, the reason and the way they were held constant is presented.

4.3. Controlled Variables

Table 1. Controlled variables, reasons for control, and the method of control.

Controlled variable	Reason for control	Method of control
Volume of yeast solution used per trial	So that the same number of yeast organisms are present in each trial	50 ml measured using a volumetric cylinder
Type of yeast (*Saccharomyces cerevisae*) used per trial	Different yeasts may carry out respiration at different rates and may be affected by UV radiation in different ways	Dry baker's yeast
Volume of Frezyderm seaside sunscreen used per trial	So that the same number of sunscreen molecules are present for each different time of exposure	9.0 ml measured using a volumetric cylinder
Number of trials per exposure to UV radiation	So that the experiment is fair and so that sufficient data can be collected	5 trials per condition

Continued

Temperature of water bath used for the control trials	As all samples which were exposed to UV radiation where at an ambient temperature of 36˚C, the control yeasts were placed in an electronic water bath at the same temperature	Water bath set at 36˚C and monitored using a thermometer
Duration that the yeast was allowed to respire for before and after UV exposure	So that the experiment is fair and so that each yeast is given the same time to respire	10 minutes each measured using a digital timer
Speed of stirring yeast solution	So that the mixing of yeast and water was at the same intensity	Magnetic stirrer set at speed number 9
Speed of stirring while yeast solution was allowed to respire	So that the mixing of yeast and water was at the same intensity	Magnetic stirrer set at speed number 2
Location of experimental setup while exposing yeast to UV radiation	To allow for the same ambient temperature and amount of UV radiation	All trials were carried out outdoors, while also checking daily for the weather, UV index, and ambient temperature (information can be found in Figures A1-A3 in **Appendix A**)

5. Materials and Apparatus

- Dry baker's yeast (*Saccharomyces cerevisiae*), 7.5 grams
- Tap water, 2000 ml
- Frezyderm Seaside sunscreen UV protection level 50+, 54 ml
- Electronic weighing scale (±0.0002 g)
- Magnetic stirrer
- Carbon dioxide sensor
- PASCO Capstone program
- Conical flask 200 ml (±25 ml)
- Volumetric cylinder 50 ml (±2.5 ml)
- Volumetric cylinder 250 ml (±10 ml)
- Beaker 50 ml (±5 ml)
- Beaker 500 ml (±50 ml)
- Spatula
- Hot plate
- Electronic water bath set at 36˚C
- Thermometer
- Digital timer (±0.1 s)
- Plastic pipette
- Glass stirring rod
- Permanent marker
- Tweezer

6. Method

6.1. Part A: Measuring Carbon Dioxide

1) 500 ml of tap water was measured using a volumetric cylinder and added to a beaker.

2) 2.5 grams of dry yeast were measured using an electronic weighing scale and added to the beaker containing the water.

3) The beaker was placed on a hot plate and a magnetic stirrer was added.

4) The stirrer was switched on at speed 9 and was allowed to stir until all the yeast had dissolved.

5) 50 ml of yeast solution was measured using a volumetric cylinder and added to a 250 ml conical flask

6) 9 ml of sunscreen was measured using a volumetric cylinder and added into the conical flask containing the 50 ml yeast forming a protective layer.

7) A small magnetic stirrer was added and switched on at speed 2.

8) The carbon dioxide sensor was placed in position at the opening of the conical flask.

9) RUN was pressed on the PASCO Capstone program and the levels of carbon dioxide (in ppm) were measured for 10 minutes.

10) The flask was then taken outdoors and sat under UV radiation for 60 minutes.

11) Steps 8 and 9 were repeated.

12) Steps 5 - 10 were repeated four more times.

13) Steps 5 - 12 were repeated for the remaining exposure times (15 min, 30 min)

14) Steps 1 - 13 were repeated in the absence of sunscreen (without step 6).

15) For the control (0 min exposure) steps 1 - 6 were repeated.

16) The conical flask was placed in an electronic water bath set at 36˚C until the mixture reached the desired temperature.

17) Steps 7 - 9 were repeated.

18) Steps 15 - 17 were repeated in the absence of sunscreen.

6.2. Part B: Measuring Number of Yeast Cells

1) 500 ml of tap water was measured using a volumetric cylinder and added to a beaker.

2) 2.5 grams of dry yeast were measured using an electronic weighing scale and added to the beaker containing the water.

3) The beaker was placed on a hot plate and a magnetic stirrer was added.

4) The stirrer was switched on at speed 9 and was allowed to stir until all the yeast had dissolved.

5) 3 ml of distilled water were measured using a plastic pipette and poured in 1 glass cuvette as the control measurement.

6) 3 ml of the yeast solution were measured using a plastic pipette and poured in 1 glass cuvette.

7) The two glass cuvettes were placed in the 2nd and 3rd slots of the spectrophotometer respectively.

8) Calibration at the distilled water took place, until it reached exactly 100%.

9) The drawer with the two cuvettes was moved and the cuvette with the solution was now in front of the light source.

10) The % mode was changed to the absorption setting.

11) Time passed until the absorbance was stable.

12) Dilutions were done until the absorbance was equal to 0.125.

13) Calculations were made to convert the absorbance into density ($N = 0.125 * r$).

14) 50 ml of yeast solution was measured using a volumetric cylinder and added to a 250 ml conical flask.

15) 9 ml of sunscreen was measured using a volumetric cylinder and added into the conical flask containing the 50 ml yeast forming a protective layer.

16) 4 conical flasks were prepared without sunscreen and 4 with sunscreen, with each the 50 ml of yeast solution.

17) One of each category of the conical flaks were the control measurements which were not exposed to the sun at all.

18) 1 of each flask was then exposed to the sun for 15 min, 30 min, 60 min.

19) After each period steps 5 - 13 were repeated.

7. Data Collection

The following Table 2 is an example of the data collected from the concentration of carbon dioxide released from the yeast cells with no exposure to UV. The remaining data can be found in the **Appendix B** (Tables B1-B15).

Table 2. Concentration of carbon dioxide released with no exposure to UV (control) **without** sunscreen.

Time of recording (min)	Concentration of carbon dioxide/ppm			
	Duration of culture growth			
	15 min	30 min	45 min	60 min
0	900	404	702	676
1.0	1620	1206	1024	1022
2.0	2118	1640	1474	1322
3.0	2970	2006	1786	1620
4.0	3574	2466	2096	1892
5.0	4114	2880	2464	2192
6.0	4624	3266	2794	2512
7.0	5084	3620	3126	2796
8.0	5604	3930	3412	3062
9.0	5994	4192	3644	3328
10.0	6520	4510	3946	3568

8. Processing of Data

In each table the rate of the change of CO_2 is calculated, per time, and then the average rate is estimated together with the standard deviation values.

1) The rate is calculated with the following formula (sample calculation, Table B1, Trial 1):

$$\text{rate of carbon dioxide concentration change} = \frac{(\text{final value} - \text{initial value})\,\text{ppm}}{10\ \text{minutes}} = \frac{(7526 - 528)\,\text{ppm}}{10\ \text{min}} = 700\,\text{ppm}\cdot\text{min}^{-1}$$

2) The average rate from the five trials is calculated with the following formula (sample calculation, Table 1, Trials 1 - 5):

$$\text{Average rate of carbon dioxide concentration change} = \left(\frac{\text{Rate triall} + \cdots + \text{Rate trial5}}{5}\right)\text{ppm}\cdot\text{min}^{-1} = 741\ \text{ppm}\cdot\text{min}^{-1}$$

3) With an SD value of **94 ppm·min^{-1}**.

These results are summed up and presented in the following Table 3.

The following figure illustrates the changes in CO_2 concentration with both the presence or absence of sunscreen over the yeast cells. The results presented in these tables are introduced to Excel to construct relevant graphs (Figures 4-7).

Table 3. Rate of CO_2 concentration change over time in different UV exposure duration and in the presence or absence of sunscreen.

Conditions	Rate of CO_2 concentration change over time/ppm·min^{-1}						
	Trials						
	1	2	3	4	5	Average	SD
0 exposure, no sunscreen	700	776	873	770	616	741	94
0 exposure, with sunscreen	1117	1105	1239	1118	1222	1160	65
Before 15 min exposure no sunscreen	531	520	476	455	485	493	32
After 15 min exposure no sunscreen	154	156	141	129	142	145	11
Before 15 min exposure with sunscreen	1212	1135	1166	1103	1272	1178	66
After 15 min exposure with sunscreen	1158	956	1035	1008	1005	1032	76
Before 30 min exposure no sunscreen	1065	906	928	952	851	904	79
After 30 min exposure no sunscreen	765	492	572	691	634	631	94
Before 30 min exposure with sunscreen	1343	1363	1475	1588	1477	1449	99
After 30 min exposure with sunscreen	1337	1342	1377	1564	1288	1381	107
Before 60 min exposure no sunscreen	702	637	599	495	473	581	97

Continued

After 60 min exposure no sunscreen	483	471	415	398	549	462	60
Before 60 min exposure with sunscreen	1146	1237	1311	1351	1273	1284	93
After 60 min exposure with sunscreen	1328	1209	1476	1227	1288	1326	92

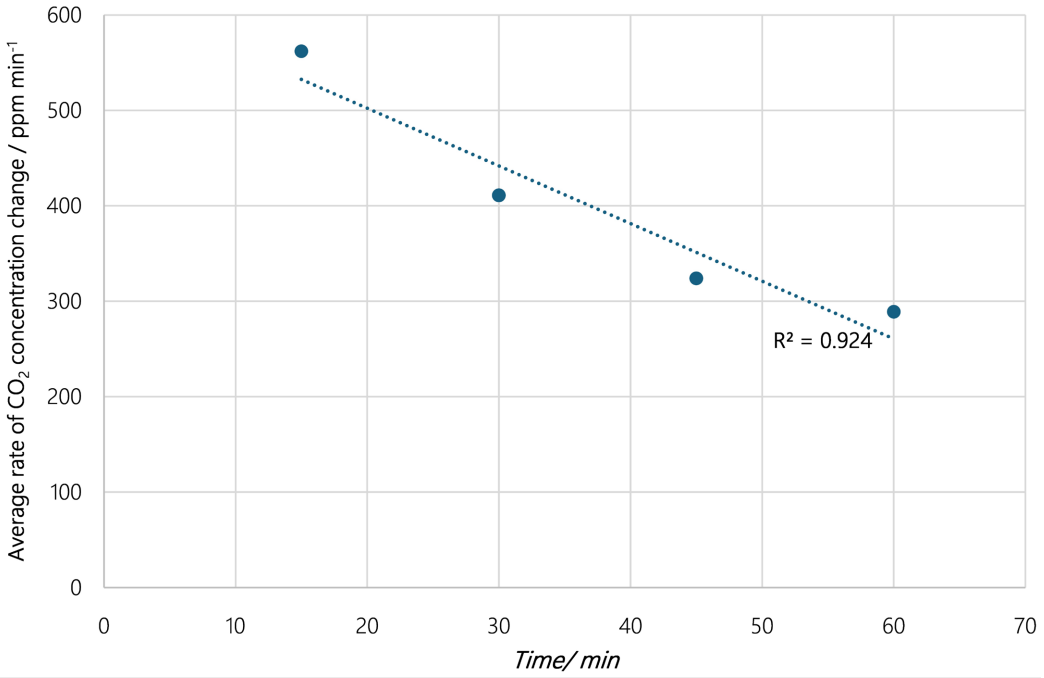

Figure 4. Concentration of carbon dioxide released with no exposure to UV (control) without sunscreen.

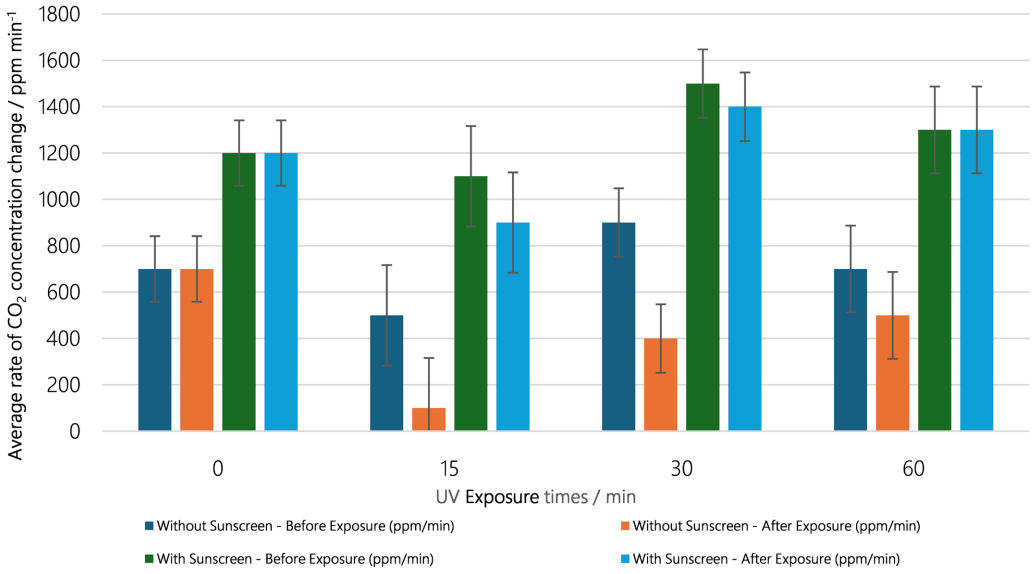

Figure 5. Average rate of carbon dioxide concentration change before and after exposure of yeast to UV for increasing times, with and without sunscreen (Error bars show ±1sd).

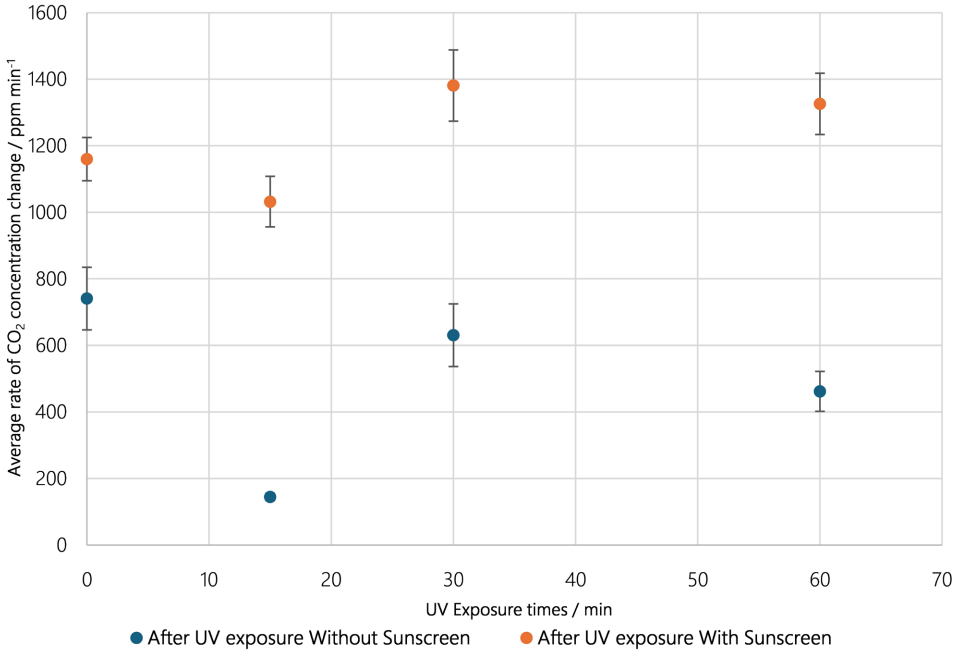

Figure 6. Average rate of carbon dioxide concentration change after exposure to UV for increasing times, with and without sunscreen (Error bars show ±1sd).

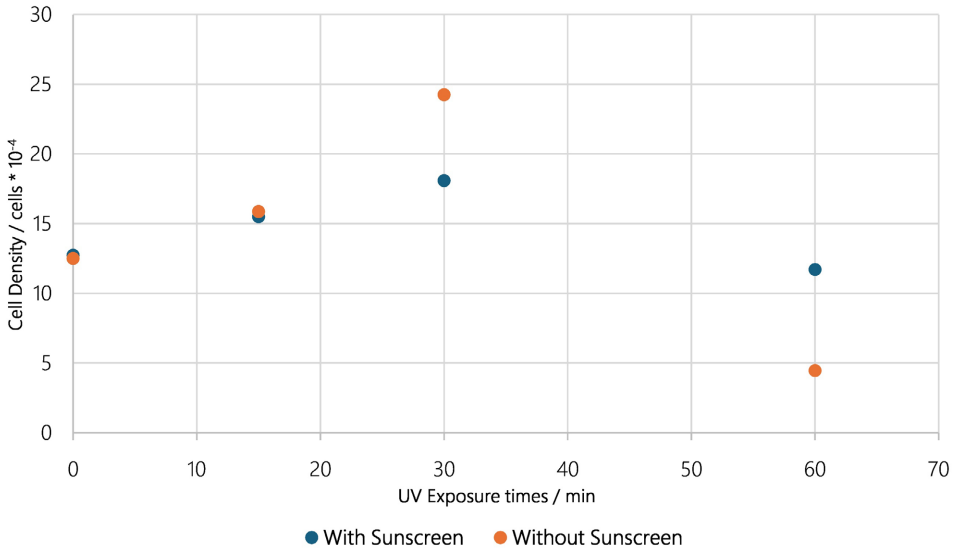

Figure 7. Cell density assessed spectrophotometrically with and without sunscreen versus times of UV exposure.

9. Statistical Analysis

1) Rate of CO_2 Concentration Change:

The t-test comparing the rates of CO_2 concentration change between the trials with sunscreen and without sunscreen shows that:

- **t-statistic**: 6.577
- **p-value**: 2.62×10^{-5}

This is a very low p-value that indicates a statistically significant difference

between the rates of CO_2 concentration change with and without sunscreen across all exposure times.

2) Cell Density:

The t-test comparing the cell densities with sunscreen and without sunscreen shows that:

- **t-statistic**: 0.055
- **p-value**: 0.958

This high p-value suggests there is no statistically significant difference in cell density between the groups with and without sunscreen.

The important finding in this case is that the sunscreen appears to have a significant effect on the rate of CO_2 production, but not on cell density.

3) To analyze whether there are significant differences before and after UV exposure both with and without sunscreen, separate t-tests were run for the before and after groups.

The paired t-test results comparing the rate of CO_2 concentration change before and after UV exposure show the following:

- **Without Sunscreen:**
  - t-statistic = 7.76
  - p-value = 1.94×10^{-6}

This p-value is much smaller than the significance level of 0.05, indicating a statistically significant difference between the rates before and after UV exposure without sunscreen.

- **With sunscreen:**
  - t-statistic = 1.81
  - p-value = 0.091

This p-value is larger than the significance level of 0.05, indicating no statistically significant difference between the rates before and after UV exposure with sunscreen.

In conclusion:

1) There is a significant change in the rate of CO_2 concentration without sunscreen after UV exposure.

2) There is no significant change in the rate with sunscreen after UV exposure, indicating sunscreen helps decrease the adverse effect of UV on respiration rates in yeast.

10. Discussion

The observed phenomena that the cell density differences are not statistically significant, while the respiration rates are, can be explained by taking into account the effects of UV radiation on cellular metabolic processes apart from cell survival. UV radiation, mostly UVB and UVC, can cause significant damage to cellular molecules like DNA, proteins, and membranes, which would detrimentally affect the cell's ability to function normally, even in the case that the cell survives.

1) Metabolic Machinery Damage: Although the overall number of yeast cells

shown by the cell density remains intact between groups with and without sunscreen, UV radiation can damage cellular molecules essential for respiration. UV radiation causes damage to DNA, proteins, and enzymes, especially those involved in respiration like cytochrome c oxidase or enzymes in the glycolytic pathway [24]. The damage of such proteins, disrupts the electron transport chain. Thus, the cells may remain alive (thus the density remains the same), but their ability to produce ATP is compromised. Moreover, damage to mitochondria or other key organelles involved in energy production can lead to reduced metabolic efficiency [25] [26]. These damages in DNA impair the yeast cells' ability to carry out oxidative phosphorylation, which happens in human cells, too.

2) Sublethal Damage: UV exposure may cause sublethal damage, where the yeast cells are not instantly killed but experience dysfunction. In such cases the cells may divide or survive but with impaired metabolic functions [27]. This may explain why the respiration rates are significantly lower without sunscreen, as UV-damaged cells struggle to perform effectively their cellular respiration [27] [28].

3) Heterogeneity of Damage: Not all cells may be equally influenced by UV radiation. Some cells in the population may be more resistant to UV, while others may be significantly damaged. This in turn could create a population where some cells continue to respire normally, whereas others would show severely decreased respiration, keeping the density constant but decreasing the overall CO_2 production [24] [29].

4) Apoptosis or Programmed Cell Death: UV radiation may also induce apoptosis or programmed cell death mechanisms in yeast, which could result in cells that remain intact, contributing to the cell density, but being metabolically inactive, therefore contributing less to respiration. In this case, cells damaged by UV can still be detected by the spectrophotometer measuring density, but they are not functioning normally in terms of metabolism [30] [31].

5) Sunscreen Protection: Sunscreen creates protective barriers that absorb, scatter, or reflect UV radiation. The ingredients that do so may be Zinc Oxide, Titanium Dioxide, and other UV filters. This way, the UV cannot penetrate the cell membrane. This membrane can be the membrane of a yeast cell, or the cell of human skin [32].

6) Relation with Human Skin Cells: It is true that yeast cells have similarities with human skin cells. Most similarities are found in the metabolic processes that both types of cells follow. Except for metabolic processes, they follow similar division and growth processes. The similarities are the reasons that yeast cells can be a model to study human cells. But, there are also differences that cause limitations. Such differences are the process of respiration without oxygen present. Yeast cells follow alcoholic fermentation, while human cells undergo anaerobic respiration.

7) General Information: Since the role of sunscreen is so major it must be spread. Schools must implement specific sessions, not only for students, but parents as well. Another way to increase awareness, and cancer prevention, is to directly provide people with sunscreens. On the other hand, there might be a

disadvantage concerning the environment. All the plastic bottles and caps, can easily be carried by wind during summer, polluting beaches and nature. This might also kill animals, distracting ecosystems [33].

By looking at the data collected we can make the following observations:

For yeast without sunscreen, the average rate of CO_2 concentration did not follow a clear trend. At 0 minutes without UV exposure, the rate was 741 ppm per minute, decreasing after 15 minutes and nearly doubling at 30 minutes, indicating peak respiration. After UV exposure, respiration began later and increased more slowly, showing yeast respired faster without UV exposure.

With sunscreen, CO_2 production was generally much higher, both with and without UV exposure. UV radiation slowed the onset of respiration, but CO_2 production at 15, 30, and 60 minutes was consistently higher. At 60 minutes, yeast exposed to UV with sunscreen respired faster than those unexposed. The small difference in average CO_2 concentration before and after exposure suggests effective protection by sunscreen. The statistical analysis supports this, with a t-statistic (6.577) and a p-value (2.62×10^{-5}) confirming sunscreen's protective role against UV-induced metabolic damage and a t-statistic (7.76) with a p-value (1.94×10^{-6}) highlighting the harmful effects of UV on yeast respiration. This aligned with the hypothesis, since without sunscreen, the yeast cells' survival decreased. This was more evident at the high exposure time (60 minutes). On the other hand, in the presence of sunscreen most yeast cells survived, with the rate of respiration increasing.

In conclusion, this study highlights the significant protective role of sunscreen in mitigating the adverse effects of UV radiation on *Saccharomyces cerevisiae*, a eukaryotic model organism. By preserving metabolic processes and shielding cells from UV-induced damage, sunscreen proves critical in protecting cellular components essential for survival. Furthermore, the findings establish a clear link between UV exposure and metabolic dysfunction, reinforcing the parallels between yeast and human skin cells and emphasizing sunscreen's importance in promoting cellular health and preventing damage.

11. Evaluation

In Table 4, the limitations of the experiment and ways to improve such problems are suggested.

Table 4. Evaluation of data collected.

Limitations	Suggested improvements
In the control runs, the water bath was used to match the yeast solution's temperature to the ambient temperature of the other samples. However, some flasks remained in the water bath longer, causing temperature variations that could have affected the cells differently.	To improve this, each trial should be carried out on its own, placing only one flask within the water bath and removing it at the appropriate time.

Continued

The effects of UV radiation were examined on only one type of yeast, dry yeast.	To improve this, repeat the procedure using wet yeast, to collect comparable data.
The location where the flasks were placed was not kept constant, due to the absence or presence of sunlight on each particular day.	A solution of this would be to place them in the UV chamber, so that they receive the same amount of radiation, however it would only be UVC.
The UV intensity varied from day to day due to the presence of clouds. (**Figure A3**, **Appendix A**)	Yeasts should be placed in the UV chamber, so that they receive the same amount of radiation, however it would only be UVC.
The magnetic stirrer was not on operation while the data was being collected.	To improve this, leave the magnetic stirrer on during the entire duration of data collection.
The flasks were left outdoors for a longer period of time than others, thus receiving more UV radiation.	To improve this, each trial should be carried out on its own, placing only one flask outdoors at each time.
Some specimens of yeast were not measured directly after the appropriate time of exposure to UV radiation, and were left indoors for some period of time.	To improve this, set up more than one stations, or carry out a larger number of trials, for example 10 trials, so as to collect more sufficient data.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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Appendix A

Table A1. Chemical composition of Frezyderm seaside sunscreen UV protection level 50+ [22].

Ingredient name	Purpose	Wavelength protection
C_{13-15} alkane	Solvent, emollient	-
Ethylhexyl Methoxycinnamate	sunscreen	UVB [34]
Dibutyl adipate	Emollient, solvent	
Diethylamino hydroxybenzyl Hexyl benzoate	sunscreen	UVA [35]
Octocrylene	sunscreen	UVA, UVB [36]
Ethylhexyl salicylate	sunscreen	UVB [37]
Butyl methoxydibenzoylmethane	sunscreen	UVA [38]
Perfume	Perfuming	-
Benzyl alcohol	Preservation, perfuming, solvent, viscosity, controlling	-

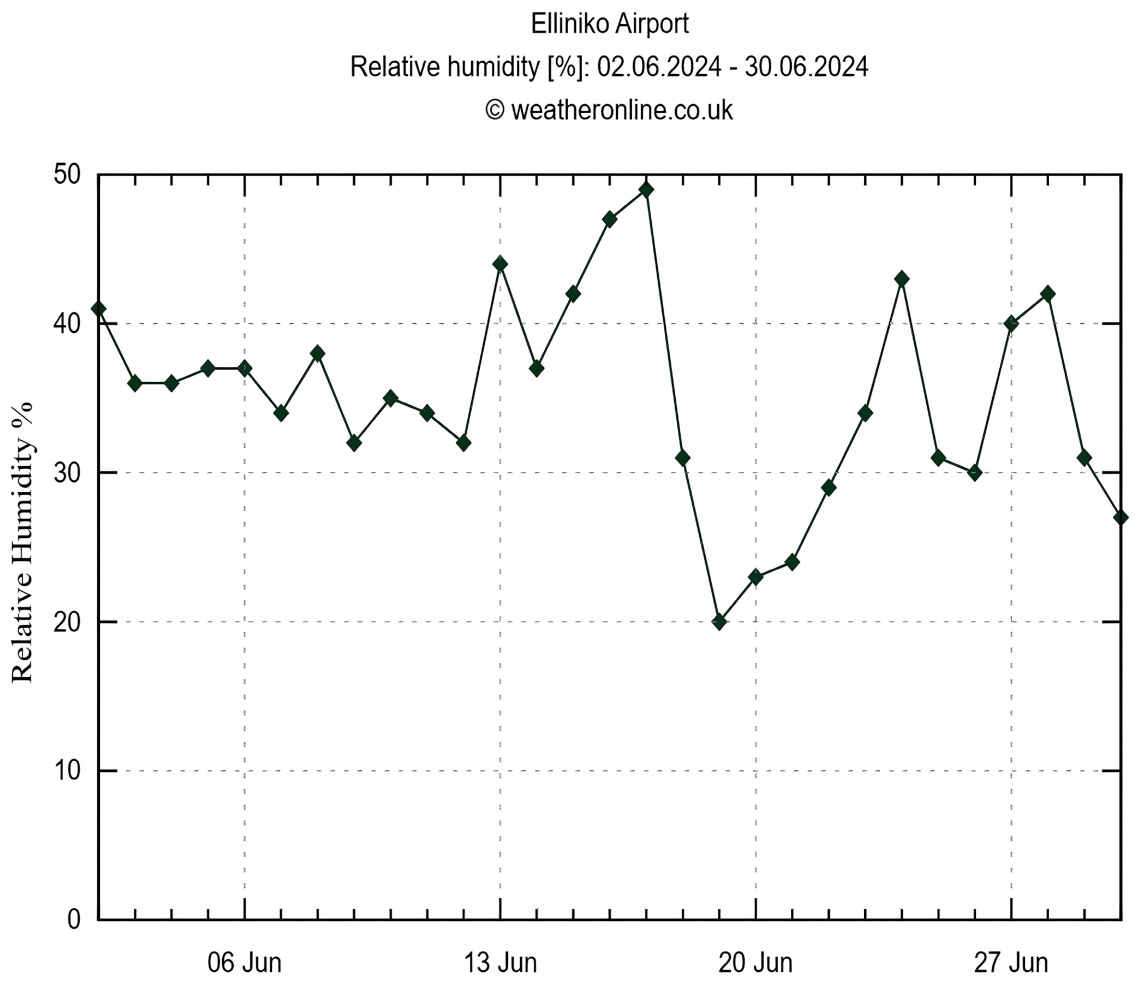

Figure A1. Relative humidity during 2 June-30 June 2024, when I carried out the experiment.

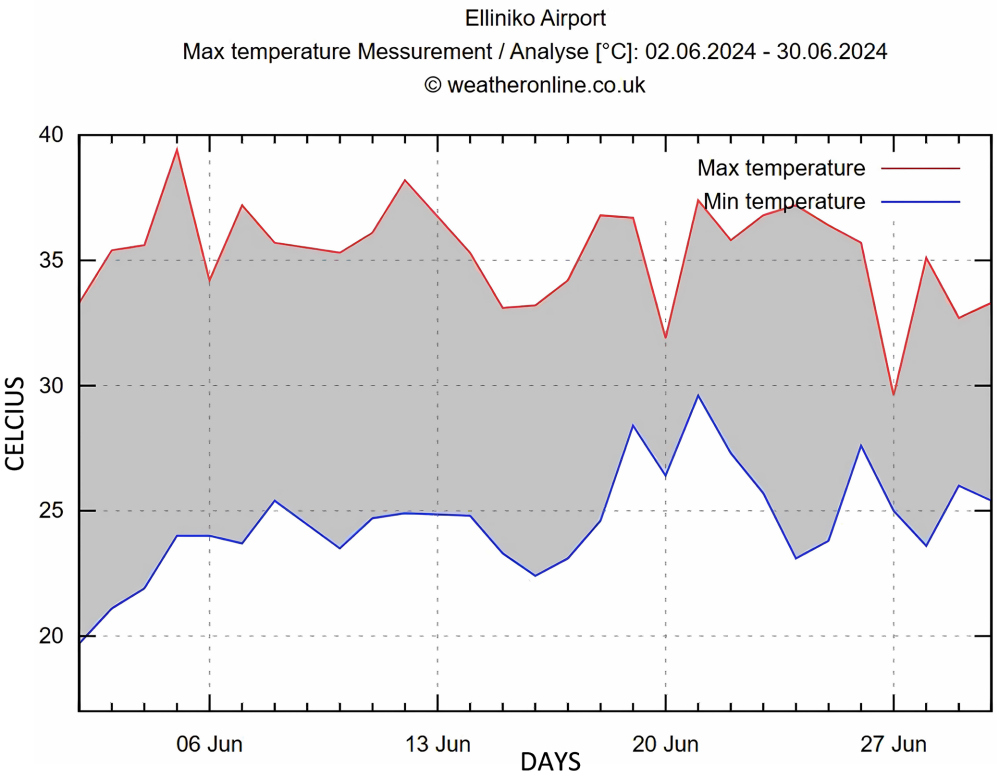

Figure A2. Maximum temperature measurements during 2 June-30 June 2024, when I carried out my experiment.

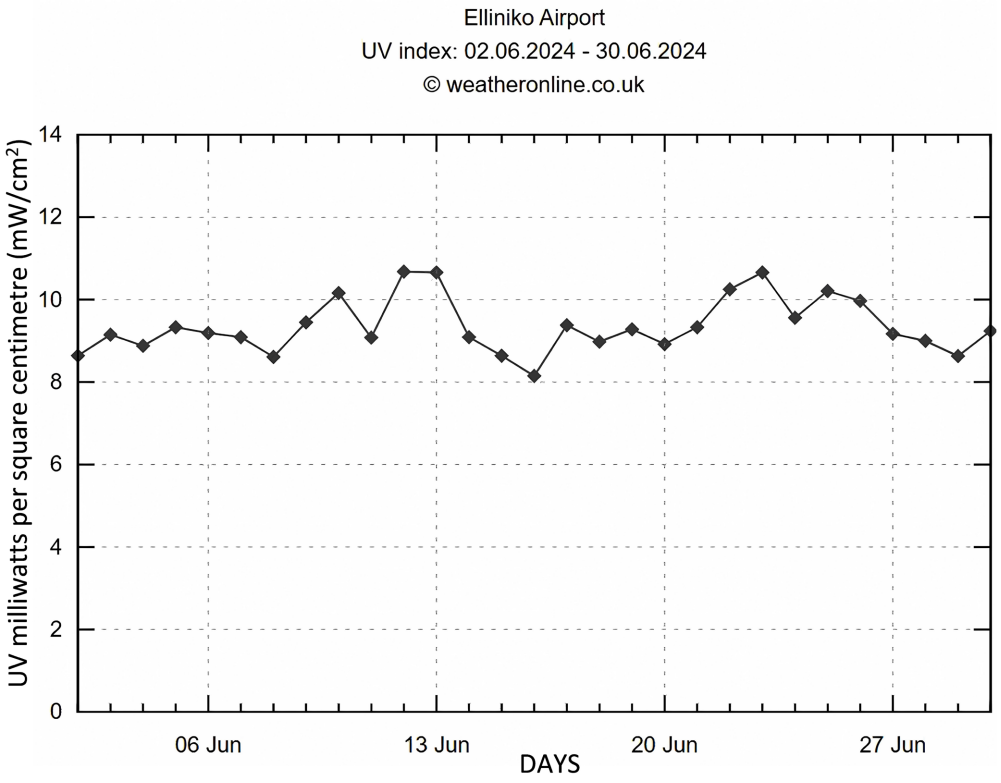

Figure A3. UV index during 2 June-30 June 2024, when I carried out my experiment.

Appendix B

Table B1. Concentration of carbon dioxide released at 0 min exposure **without** sunscreen.

Control	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	528	922	582	826	956
1.0	1686	2214	1794	1774	1770
2.0	2488	3290	3032	2864	2740
3.0	3332	4232	4118	3814	3532
4.0	4058	4976	4980	4598	4194
5.0	4706	5788	5842	5408	4750
6.0	5334	6478	6648	6200	5300
7.0	5962	7074	7414	6892	5792
8.0	6532	7612	8100	7480	6216
9.0	7058	8092	8744	8000	6610
10.0	7526	8682	9316	8522	7116

Table B2. Concentration of carbon dioxide released at 0 min exposure **with** sunscreen.

Control	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	1238	1208	4244	1776	4496
1.0	1980	2052	5370	1818	5482
2.0	3442	3642	6026	3106	6462
3.0	4716	4924	7704	4410	8060
4.0	6124	6248	9172	5620	10,064
5.0	7350	7360	11,366	7006	11,920
6.0	8488	8402	13,072	8364	13,354
7.0	9492	9352	14,386	9556	14,502
8.0	10,472	10,196	15,294	10,734	15,516
9.0	11,482	11,136	16,034	11,764	16,214
10.0	12,406	12,260	16,632	12,956	16,718

Table B3. Concentration of carbon dioxide released before 15 min exposure **without** sunscreen.

Before	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	528	982	664	742	846
1.0	1200	1346	1192	1012	1190
2.0	1608	1748	1532	1354	1576
3.0	2158	2214	2028	1868	1990

Continued

4.0	2782	2860	2596	2416	2588
5.0	3364	3492	3096	2934	3182
6.0	3916	4086	3560	3430	3738
7.0	4456	4650	4082	3922	4284
8.0	4940	5188	4560	4398	4786
9.0	5408	5692	5004	4854	5264
10.0	5842	6178	5426	5288	5700

Table B4. Concentration of carbon dioxide released after 15 min exposure **without** sunscreen.

After	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	500	600	550	480	510
1.0	764	854	792	728	770
2.0	814	902	840	780	818
3.0	844	934	872	812	848
4.0	874	966	904	838	872
5.0	898	992	926	864	894
6.0	926	1012	950	888	918
7.0	1096	1194	1064	994	1040
8.0	1432	1504	1348	1240	1344
9.0	1738	1816	1654	1502	1648
10.0	2044	2156	1964	1774	1934

Table B5. Concentration of carbon dioxide released before 15 min exposure **with** sunscreen.

Before	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	564	1012	944	550	3538
1.0	912	1532	1572	1200	4372
2.0	1562	2340	3058	2954	5470
3.0	2328	4144	4386	3030	6506
4.0	3194	5282	5654	4240	8230
5.0	4970	6596	6954	5500	9836
6.0	5682	7642	8118	6768	11,550
7.0	6430	8618	9294	7922	13,072
8.0	7212	9526	10,428	8066	14,348
9.0	9970	10,380	11,570	9504	15,388
10.0	12,688	12,366	12,602	11,586	16,260

Table B6. Concentration of carbon dioxide released after 15 min exposure **with** sunscreen.

After	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	1318	1030	1132	1024	1262
1.0	2416	1832	2156	2394	2250
2.0	4136	2878	3654	3800	3806
3.0	5838	4026	4966	4972	5218
4.0	7326	5206	6326	6196	6572
5.0	8512	6428	7450	7266	7622
6.0	9526	7454	8350	8214	8532
7.0	10,534	8360	9220	9098	9330
8.0	11,430	9160	9966	9784	10,000
9.0	12,240	9828	10,762	10,442	10,712
10.0	12,900	10,590	11,486	11,106	11,308

Table B7. Concentration of carbon dioxide released before 30 min exposure **without** sunscreen.

Before	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	1740	1694	1756	1588	878
1.0	2922	2890	2744	2954	1652
2.0	5354	4696	4818	4736	3346
3.0	7164	6158	6356	6250	4680
4.0	8394	7216	7472	7412	5734
5.0	9208	8006	8268	8266	6582
6.0	9824	8680	8884	8946	7250
7.0	10,510	9224	9432	9502	7800
8.0	11,234	9728	9916	9986	8346
9.0	11,878	10,246	10,488	10,566	8896
10.0	12,390	10,756	11,036	11,108	9386

Table B8. Concentration of carbon dioxide released after 30 min exposure **without** sunscreen.

After	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	500	1544	1342	938	916
1.0	2298	1124	1816	1956	2210
2.0	3926	1108	3102	3478	3454
3.0	4910	1780	4096	4474	4342

Continued

4.0	5714	2974	4746	5196	4952
5.0	6254	3938	5272	5874	5486
6.0	6558	4620	5766	6384	5908
7.0	6980	5162	6122	6828	6272
8.0	7414	5672	6464	7212	6622
9.0	7798	6092	6792	7540	6968
10.0	8148	6466	7060	7844	7256

Table B9. Concentration of carbon dioxide released before 30 min exposure **with** sunscreen.

Before	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	1686	1478	1936	5874	4380
1.0	2210	2806	2178	7342	5228
2.0	4642	4328	4864	8668	6516
3.0	6900	5832	7210	9600	8764
4.0	8662	7340	8956	12,518	9714
5.0	10,116	8688	10,296	14,824	12,776
6.0	11,616	9844	11,616	16,546	14,502
7.0	12,770	11,148	13,620	17,876	15,776
8.0	13,682	12,360	14,400	18,930	17,120
9.0	14,440	14,278	15,060	19,814	18,112
10.0	15,120	15,106	16,690	21,750	19,152

Table B10. Concentration of carbon dioxide released after 30 min exposure **with** sunscreen.

After	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	2350	2662	2420	2440	3400
1.0	3298	3560	3812	3748	4102
2.0	4564	4889	5186	5772	5606
3.0	6090	6410	6930	8250	6898
4.0	7744	8054	8542	10,900	8994
5.0	9442	9762	10,266	12,820	10,854
6.0	11,036	11,290	11,562	14,354	12,466
7.0	12,450	12,778	12,820	15,586	13,640
8.0	13,710	13,050	13,980	16,540	14,672
9.0	14,784	15,043	15,106	17,264	15,510
10.0	15,720	16,080	16,182	18,080	16,284

Table B11. Concentration of carbon dioxide released before 60 min exposure **without** sunscreen.

Before	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	640	1274	724	696	814
1.0	1476	1894	1144	1066	1066
2.0	2180	2690	1866	1574	1486
3.0	3148	3476	2682	2132	1946
4.0	3940	4202	3412	2782	2534
5.0	4630	4872	4050	3344	3116
6.0	5300	5564	4614	3862	3634
7.0	5992	6184	5160	4352	4138
8.0	6604	6726	5740	4790	4594
9.0	7160	7212	6254	5202	5034
10.0	7660	7648	6710	5644	5542

Table B12. Concentration of carbon dioxide released after 60 min exposure **without** sunscreen.

After	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	500	438	622	1288	810
1.0	1124	1032	894	1714	972
2.0	1758	1572	1320	2256	1728
3.0	2392	2032	1834	2796	2502
4.0	2984	2604	2376	3276	3256
5.0	3444	3130	2892	3732	3880
6.0	3904	3606	3336	4120	4436
7.0	4310	4050	3758	4476	4948
8.0	4674	4420	4122	4796	5502
9.0	5002	4776	4462	5122	6028
10.0	5332	5146	4770	5268	6302

Table B13. Concentration of carbon dioxide released before 60 min exposure **with** sunscreen.

Before	Concentration of carbon dioxide/ppm				
Time (min)	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
0	530	550	1144	1828	2374
1.0	1314	1740	2092	2370	2808
2.0	2118	3660	4198	4464	5472

Continued

3.0	3172	5262	5898	6792	7858
4.0	4244	6622	7478	8650	9722
5.0	5256	7746	8892	10,236	11,364
6.0	6334	8824	10,072	11,778	12,892
7.0	7232	9962	11,368	12,884	14,054
8.0	8074	10,886	12,468	13,804	14,926
9.0	9844	11,812	13,336	14,658	15,808
10.0	11,992	12,916	14,250	15,334	16,116

Table B14. Concentration of carbon dioxide released after 60 min exposure **with** sunscreen.

After	Concentration of carbon dioxide/ppm				
Time (min)	**Trial 1**	**Trial 2**	**Trial 3**	**Trial 4**	**Trial 5**
0	6582	5422	2572	4608	1856
1.0	7268	6076	4882	6558	3824
2.0	8174	7160	8042	7938	6270
3.0	9784	9622	10,640	10,118	8160
4.0	12,730	11,854	12,666	12,212	9712
5.0	12,750	13,712	14,166	13,766	11,164
6.0	16,206	15,088	15,162	14,774	12,392
7.0	17,444	16,132	15,972	15,464	13,334
8.0	18,432	17,036	16,648	16,118	13,902
9.0	19,204	17,870	16,994	16,468	14,314
10.0	19,869	18,508	17,328	16,882	14,738

Table B15. Cell density with and without sunscreen for each exposure time.

Before exposure: $N = 18.92 \times 10^{-4}$		
Time of exposure in UV (min)	**With sunscreen (9.0 ml/50)**	**Without sunscreen**
Control	12.73×10^{-4}	12.5×10^{-4}
15	15.5×10^{-4}	15.86×10^{-4}
30	18.08×10^{-4}	24.24×10^{-4}
60	11.71×10^{-4}	4.46×10^{-4}