

EFFECT OF SUNBATH ON AUTONOMIC VARIABLES IN HYPERTENSIVE INDIVIDUALS

¹*Dr. Deeksha Shenoy G., ²Dr. Ganesh Prasad B., ³Dr. Prashanth Shetty, ⁴Ananth Prabhu and ⁵Dr. Sujatha K. J.

¹M.D. (Clinical Naturopathy) Post Graduate, Department of Naturopathy clinical, SDM College of Naturopathy & Yogic sciences, Ujire, Karnataka, India.

²Assistant Professor, Department of Fasting & Dietetics SDM College of Naturopathy and Yogic Sciences, Ujire, Karnataka, India.

³Principal, SDM College of Naturopathy and Yogic Sciences, Ujire, Karnataka, India.

⁴Assistant Professor, Department of Pharmaceutics, NGS Institute of Pharmaceutical Sciences, Deralakatte, Mangalore, Karnataka, India.

⁵Dean, Division of Natural Therapeutics, Department Of Post Graduate Studies, SDM College Of Naturopathy And Yogic Sciences.

*Corresponding Author: Dr. Deeksha Shenoy G.

M.D. (Clinical Naturopathy) Post Graduate, Department of Naturopathy clinical, SDM College of Naturopathy & Yogic sciences, Ujire, Karnataka, India.

Article Received on 10/03/2020

Article Revised on 30/03/2020

Article Accepted on 20/04/2020

ABSTRACT

Background & Objectives: Sunbath is a cost-effective, efficient treatment modality used in the field of Naturopathy. Sunbath produces different physiological responses among individuals. Seasonal variations in blood pressure and cardiovascular mortality and morbidity are always encountered across the globe. Ultraviolet component of sunrays shows significant benefits on Blood Pressure. Present study aims to evaluate the effect of sunbath on various autonomic variables, to substantiate the clinical understanding of its effect, and to know its role in clinical application on hypertensive individuals. **Methods:** 60 subjects with hypertension between 30 to 50 years (40.08 ± 5.58) were recruited, and were randomly allocated into case (Sunbath) and control groups (without sunbath). Blood Pressure, Heart rate, Heart Rate Variability, Respiratory Rate, Digit pulse volume, Body temperatures were assessed before, immediately after and an hour after the insolation. **Results:** The data analysis showed significant reduction in SBP ($p=0.0060$), DBP ($p=0.0002$) and MAP ($p=0.0002$). In time domain of HRV, there was significant increase in Mean RR ($p=0.0052$), RMSSD ($p=0.0039$), NN50 ($p=0.0042$), pNN50 ($p<0.0001$) and significant reduction in HR ($p=0.0005$) were noted. In frequency domain, significant increase in HF ($p=0.0049$) and a significant decrease in VLF ($p=0.0003$), LF ($p=0.0001$) and LF/HF ratio ($p=0.0013$) were noted. Even after 1 hour, changes were sustained. No significant changes were observed in Pulse rate and Temperature. **Interpretation & Conclusion:** The present study reported that sunbath reduces the sympathetic tone and shifts the sympathovagal balance in favor of parasympathetic dominance and claims that it can be effectively used in managing hypertension.

KEYWORDS: Sunbath; Heart rate; Parasympathetic activity; Autonomic Nervous System; Hypertension.

INTRODUCTION

Hypertension is one of the important preventable non-communicable diseases (NCD), which globally accounts for premature death and disability.^[1] It is clinically classified as primary and secondary hypertension.^[2] Many interrelated factors do contribute to the raised blood pressure and those includes-salt sensitivity, cardiac output and peripheral vascular resistance, obesity and insulin resistance, the renin-angiotensin-aldosterone system, the sympathetic nervous system, bradykinin and endothelin, endothelial derived relaxing factor(nitric oxide), atrial natriuretic peptide, also the other well evaluated factors such as genetics, endothelial

dysfunction, low birth weight and intrauterine nutrition, and also neurovascular anomalies.^[3] Older age, overweight, unhealthy diet, lack of physical exercise, psychological factors, smoking tobacco products and family history of hypertension also acts as major predisposing factors towards the causation of hypertension.

Uncontrolled hypertension escalates the risks of end organ damage and complications, such as left ventricular hypertrophy, diastolic dysfunction, congestive heart failure, ischemic heart disease, strokes and end stage renal failure. Appropriate management of hypertension

still remains as a challenge and is necessary to combat the increasing burden.

It is amenable to manage hypertension through both non-pharmacological lifestyle modification and medications. The primary preventive measures should always be considered first towards the management of hypertension, and that includes life style changes, dietary modifications, physical exercise and weight management.^[4] In conventional medicine, the primary and the secondary antihypertensive agents are used to revert the blood pressure to safer range. Despite recent advances in drug therapy, in majority of diagnosed hypertensive patients the blood pressure is poorly controlled mainly due to nonadherence to the treatment protocols.^[5]

Hence to overcome this, non-pharmacological interventions such as complementary and alternative medicine (CAM) can be efficiently employed, with its fundamental principle of being 'natural' and also because of its safety and cost effectiveness.^[6,7]

Naturopathy, an alternative medicine system is one among the five major categories of CAM. Naturopathic treatment modalities are proved to be effective in improving health outcomes and improving quality of life (QOL) in patients with or those at risk for chronic conditions.^[8] Naturopathic treatments are mainly based on the five great elements or Panchamahabhutas;^[9] namely air, water, earth, fire and ether. Sunbath (Heliotherapy) is one of the principle therapies used in naturopathy. Here Sun, the core component of fire element is used therapeutically, by exposing the body to sunlight for appropriate period of time.^[10] Therapeutically sunlight can be used in various forms to get desired physiological effects.

It has been already proven that the cardiovascular health varies with season, weather, and climate.^[11,12] Ultra violet radiation (UVR), present in sunlight has cardio protective activity through various mechanisms. Prevalence of hypertension also increases with the distance from the equator and seasonal and latitude variations are also seen in the incidence of acute coronary syndrome, stroke, and cerebrovascular diseases.^[13] Numerous studies have demonstrated that blood pressure varies inversely with ambient sunshine (insolation).^[14,15]

It is well established that hypertension is associated with autonomic dysregulation, and that can be quantitatively assessed by Heart Rate Variability (HRV) index, as it is a simple, non-invasive and yet a reliable tool for the quantitative assessment of the cardiac autonomic function.^[16]

Hence this study aims at understanding the effect of sunbath on autonomic variables of hypertensive

individuals and thereby establishing the extent of efficacy of sunbath in the management of hypertension.

METHODOLOGY

Participants

The study was conducted in Nature cure hospital in Dakshina Kannada District, Karnataka. The study included 60 pre diagnosed hypertensive individuals. Both males and females belonging to the age group between 30-50 years (40.08±5.58) were recruited for the study. Exclusion criteria for the study included subjects with secondary hypertension and with other comorbidities, female subjects during their menstruation,^[17] subjects with the history of photosensitivity and also those with substance abuse.

Subjects recruited were explained about the purpose of the study, intervention and assessment techniques and also their rights as research subjects. A signed informed consent form was obtained from each individual. Ethical clearance was also obtained from the institutional ethics committee. The subjects were assigned to their respective groups based on the randomization schedule.

Study design: A prospective randomized control study, which was designed to evaluate the effect of sun bath on autonomic variables in hypertensive individuals. Subjects were assessed before, immediately after and also an hour after the intervention.

Assessments

Subjects included in the study were assessed for blood pressure, autonomic variables, pulse rate and body temperature respectively before, immediately after and an hour after the intervention period.

The blood pressure was recorded in quiet and calm environment using clinically validated^[18] OMRON HEM-7120 Digital Blood Pressure monitor with the oscillometric principle. The ECG was assessed using MP 36 data acquisition system (BIOPAC, Montana, USA; model No: BSL 4.0 MP 36) by placing the Ag/AgCl pre gelled electrodes (Schwartz Biotech, Gujarat) according to the standard bipolar limb lead II configuration and an AC amplifier with 1.5 Hz high pass filter and 75 Hz low pass filter setting was used for the purpose. The ECG was digitized using a 12-bit analogue-to-digital converter (ADC). The data recorded was visually inspected off-line and only noise free data was included for analysis. Digit Pulse Volume was assessed using finger plethysmogram by placing the transducer on the volar surface of the distal phalanx of the left thumb.^[19] The body temperature was recorded with a mercury-in-glass thermometer by placing it under left axillary area.^[20]

Intervention

Individuals who were assigned for the case group were given sunbath. Sun bath was given in a well featured room (approximately 25 feet wide, 10 to 11 feet height), which was airy and private. Individual who is taking the

sunbath will be with minimum dressing and will expose the body parts to ambient sunshine. They were allowed to place themselves in any comfortable position like sitting, lying down and also they were allowed to walk around in the room during sunbathing.^[21] Duration of sunbath was 30 minutes (9.00 am to 9.30 am). Proper hydration was maintained by asking individual to have 1-2 glass of cold water before entering into the procedure. Head was also covered with a wet wrap.^[21]

Individuals who are assigned into the control group were placed in a room where the normal room temperature was maintained, here they were not exposed to sunlight, and instead they were guided regarding the life style modification, measures to reduce the stress, healthy dietary habits and also about the importance of physical exercises and yoga.

Data extraction

The data was collected as self-reported observations using outcome variables. The assessments were done at the beginning of the intervention (baseline data), immediately after the intervention (post assessment data 1) and also 1 hour after the intervention (post assessment data 2). The data was organized in Microsoft Excel Sheets (Version 2010).

- a) **Blood Pressure:** was measured using Digital Blood Pressure monitor (OMRON HEM-7120). The systolic and diastolic pressures were immediately recorded.
- b) **Autonomic and Respiratory Variables:**
 - **Heart Rate and Heart Rate Variability:** From the digitized ECG data, the R waves were detected to obtain a point event series of successive R-R Intervals, from which the beat to beat heart rate series were computed. Only noise free data was included for analysis by off-line visual inspection of it. Data was averaged for each 5 minute block period.

The HRV power spectrum was obtained using Fast Fourier Transform analysis (FFT) using the software Kubios HRV.^[22] Frequency domain and time domain components were analyzed separately.

In Frequency domain analysis, the energy in the HRV series of the following specific bands were studied viz. the very low frequency component (0.0-0.05 Hz), low frequency component (0.05-0.15 Hz), and high frequency component (0.15-0.50 Hz). The low frequency and high frequency values were expressed as normalized units. LF: HF was also calculated.

In Time domain analysis, following components were obtained:

- The mean R-R Interval (the mean of the intervals between adjacent QRS complexes or the instantaneous heart rate).

- RMSSD (the square root of the mean of the sum of the squares of differences between adjacent NN intervals)
 - NN50 (the number of interval differences of successive normal to normal intervals greater than 50ms)
 - pNN50 (the proportion derived by dividing NN50 by the total number of NN intervals)
- c) **Digit Pulse Volume (DPV):** The amplitude of the Digit Pulse Volume was sampled from the peak of pulse wave at 30-second intervals and was converted into millivolts.
 - d) **Body Temperature:** The body temperature was measured in axillary region and expressed in terms of Celsius.

Statistical analysis

Statistical analysis was done using Graph pad prism 6.0. Data were checked for normality using Kolmogorov-Smirnov test and analyzed using ANOVA and comparative analysis was also done between the case and control groups. For all the analysis p value less than 0.05 was accepted as an indicator for significance.

RESULTS

Immediately following Sunbath, there was a significant decrease in SBP ($p=0.0060$), DBP ($p=0.0002$) and MAP ($p=0.0002$). In time domain of HRV, there was a significant increase in Mean RR ($p=0.0052$), RMSSD ($p=0.0039$), NN50 ($p=0.0042$), pNN50 ($p < 0.0001$) and a significant reduction in HR ($p=0.0005$). In frequency domain of HRV, there was a significant increase in HF ($p=0.0049$) and a significant decrease in VLF ($p=0.0003$), LF ($p=0.0001$) and LF/HF ratio ($p=0.0013$). No significant changes were observed in Digit pulse volume & Temperature. At the other time interval, i.e. 1 hour after sunbath the changes that have been observed immediately following Sunbath were sustained. Even here no significant changes were observed in Digit Pulse volume & Temperature.

In control group, insignificant increase in SBP, MAP and also significant increase in DBP ($p=0.0477$) were observed. In time and frequency domains of HRV the changes observed were all insignificant. At other time interval also, i.e. 1 hour after sunbath insignificant changes in time and frequency domains of HRV were observed.

When case and control groups were compared with each other at different time intervals like immediately after sunbath and after 1 hour of sunbath, there was a significant difference seen in post values of SBP, DBP and MAP. There by significant differences were also observed in post values of all the variables of time domain and frequency domains of HRV except NN50 of time domain. Pulse Rate and Temperature have showed insignificant changes.

Table 1: Comparison of Pre, Post and after 1 hour values within case group.

| S. No. | Variables | Mean pre \pm SD | SEM | Mean Post \pm SD | SEM | Mean after 1 hour \pm SD | SEM | P Value |
|--------|-------------|-------------------|-------|--------------------|-------|----------------------------|-------|------------|
| 1 | SBP | 151.1 \pm 5.47 | 0.99 | 147.7 \pm 4.97 | 0.90 | 146.9 \pm 5.16 | 0.94 | 0.0060** ↓ |
| 2 | DBP | 99.73 \pm 4.51 | 0.82 | 95.60 \pm 4.53 | 0.82 | 95.13 \pm 4.41 | 0.80 | 0.0002** ↓ |
| 3 | MAP | 116.8 \pm 4.62 | 0.84 | 112.7 \pm 4.37 | 0.79 | 112.4 \pm 4.38 | 0.80 | 0.0002** ↓ |
| 4 | RR | 777.2 \pm 106.1 | 19.36 | 832.1 \pm 113.6 | 18.04 | 832.6 \pm 113.7 | 20.76 | 0.0052** ↑ |
| 5 | HR | 79.32 \pm 10.21 | 1.86 | 70.13 \pm 10.12 | 1.84 | 70.11 \pm 10.11 | 1.84 | 0.0005** ↓ |
| 6 | RMSSD | 32.89 \pm 17.76 | 3.24 | 46.80 \pm 18.46 | 3.37 | 47.04 \pm 18.47 | 3.37 | 0.0039** ↑ |
| 7 | NN50 | 25.53 \pm 25.44 | 4.64 | 55.86 \pm 45.68 | 8.34 | 56.54 \pm 45.80 | 8.36 | 0.0042** ↑ |
| 8 | PNN50 | 6.510 \pm 6.75 | 1.23 | 16.88 \pm 11.53 | 2.10 | 17.02 \pm 11.54 | 2.10 | <0.0001**↑ |
| 9 | VLF | 43.79 \pm 17.04 | 3.11 | 30.13 \pm 13.12 | 2.39 | 29.84 \pm 13.03 | 2.37 | 0.0003** ↓ |
| 10 | LF | 55.20 \pm 15.31 | 2.79 | 41.51 \pm 13.50 | 2.46 | 40.73 \pm 13.50 | 2.46 | 0.0001** ↓ |
| 11 | HF | 45.74 \pm 14.54 | 2.65 | 56.04 \pm 13.36 | 2.43 | 56.16 \pm 13.35 | 2.43 | 0.0049** ↑ |
| 12 | LF/HF | 1.454 \pm 1.11 | 0.20 | 0.647 \pm 0.43 | 0.07 | 0.622 \pm 0.421 | 0.076 | 0.0013** ↓ |
| 13 | Pulse Rate | 78.96 \pm 6.67 | 1.21 | 81.13 \pm 6.95 | 1.26 | 80.48 \pm 6.575 | 1.200 | 0.4406 ↑ |
| 14 | Temperature | 36.63 \pm 0.459 | 0.08 | 36.78 \pm 0.43 | 0.078 | 36.66 \pm 0.49 | 0.090 | 0.4224 ↑ |

Table 2: Comparison of pre, post and after 1 hour values within control group.

| S. No. | Variables | Mean pre \pm SD | SEM | Mean Post \pm SD | SEM | Mean after 1 hour \pm SD | SEM | P Value |
|--------|-------------|-------------------|-------|--------------------|-------|----------------------------|-------|---------|
| 1 | SBP | 149.5 \pm 5.34 | 0.97 | 151.3 \pm 4.01 | 0.73 | 151.3 \pm 4.315 | 0.78 | 0.2466 |
| 2 | DBP | 97.93 \pm 3.91 | 0.71 | 100.5 \pm 3.98 | 0.72 | 99.93 \pm 4.441 | 0.81 | 0.0477* |
| 3 | MAP | 115.1 \pm 4.12 | 0.75 | 115.8 \pm 1.34 | 0.24 | 117.1 \pm 3.874 | 0.70 | 0.5567 |
| 4 | RR | 766.5 \pm 105.3 | 19.22 | 771.3 \pm 96.93 | 17.7 | 771.4 \pm 96.88 | 17.69 | 0.9770 |
| 5 | HR | 79.76 \pm 10.44 | 1.90 | 79.20 \pm 9.22 | 1.68 | 79.17 \pm 9.163 | 1.67 | 0.7272 |
| 6 | RMSSD | 39.63 \pm 19.92 | 3.63 | 31.45 \pm 22.43 | 4.09 | 31.62 \pm 22.48 | 4.10 | 0.2523 |
| 7 | NN50 | 33.77 \pm 30.88 | 5.63 | 38.73 \pm 44.09 | 8.04 | 39.13 \pm 44.35 | 8.09 | 0.8478 |
| 8 | PNN50 | 10.65 \pm 6.60 | 1.20 | 9.147 \pm 8.37 | 1.53 | 9.169 \pm 8.419 | 1.53 | 0.1714 |
| 9 | VLF | 44.93 \pm 16.41 | 2.99 | 46.45 \pm 15.28 | 2.79 | 46.46 \pm 15.31 | 2.79 | 0.9094 |
| 10 | LF | 52.06 \pm 18.10 | 3.30 | 57.33 \pm 22.62 | 4.13 | 57.34 \pm 22.75 | 4.15 | 0.5431 |
| 11 | HF | 48.25 \pm 18.64 | 3.40 | 42.06 \pm 22.05 | 4.02 | 41.92 \pm 22.08 | 4.03 | 0.4142 |
| 12 | LF/HF | 1.28 \pm 0.71 | 0.13 | 2.08 \pm 1.68 | 0.30 | 2.101 \pm 1.705 | 0.31 | 0.6254 |
| 13 | Pulse Rate | 75.68 \pm 5.62 | 1.02 | 76.76 \pm 6.11 | 1.11 | 76.73 \pm 5.750 | 1.05 | 0.7172 |
| 14 | Temperature | 36.39 \pm 0.45 | 0.08 | 36.44 \pm 0.50 | 0.092 | 36.42 \pm 0.4930 | 0.09 | 0.9398 |

*Represents significant values (*p<0.05), ** represents highly significant values (**p <0.01), SD=Standard deviation, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, HR=Heart rate, Mean RR= Mean of R-R interval, RMSSD=The square root of the mean squared difference between adjacent N-N intervals, NN50=Consecutive normal sinus (NN) intervals exceeds 50ms, pNN50=The fraction of consecutive NN intervals that differ by more than 50ms, VLF=Very low frequency power, LF=Low frequency Power, HF=High frequency power and LF/HF=Low frequency/High frequency ratio, ↓- Decrease, ↑- Increase.

DISCUSSION

Life on earth is unimaginable without the Sun. Sunlight is a portion of electromagnetic radiation given off by the Sun, UV component of sunlight has helped to evolve life.^[23] Number of studies have shown that geomagnetic and solar influences affect a wide range of health outcomes and human behavioral aspects. Exposure to simulated sunlight that contains both UVB and UVA radiations reduces cardiovascular risk factors and improves quality of life.^[24] Epidemiological data shows that sunlight exposure reduces all-cause and cardiovascular mortality.^[25, 26] WHO has reported that a significantly larger annual disease burden of about 3.3 billion DALYs worldwide is resulted from very low

levels of exposure to UV radiations.^[27] Cardiovascular health varies with season, weather, and climate. Blood pressure will also be generally high in the months with fewer hours of light.^[28,29]

Studies have suggested that there will be reduction of either systolic or diastolic or both the aspects of blood pressure following insolation.^[30,31] In our study, a significant reduction was observed in SBP, DBP and HR in the case group where sunbath was incorporated. And this indicates the parasympathetic dominance. This was further evidenced by the findings observed in other components of HRV such as a significant reduction in LF component, LF/ HF ratio and a significant increase in HF component, RMSSD, Mean RR and NN50 which are

suggestive of a shift in sympathovagal balance towards parasympathetic dominance.

Nitric oxide (NO) an endothelium derived relaxing factor is a key vasodilator in the vascular system.^[32] Photolabile nitric oxide derivatives like nitrite and S-nitroso thiols, which are present in human skin will decompose and lead to the formation of vasoactive NO after irradiation with the UVA component of sunlight.^[33] Nitric oxide synthase isoforms (Nitric oxide, Nitrite, Nitrate) which are expressed in the dermis and epidermis, can also be mobilized by sunlight and delivered to the systemic circulation to exert coronary vasodilator and cardio protective as well as antihypertensive effects.^[34] UVA irradiation enhances the release of cutaneous NO stores, 'photo relaxes' the blood vessels and also leads to peripheral vasodilatation. Photolysed circulating nitrate by UVA radiation, will contribute to light-induced blood pressure reduction and cardio protection and in turn has beneficial effects on cardiovascular mortality.^[35] Suschek and co-authors demonstrated that irradiation of healthy individuals with biologically relevant doses of UVA leads to a sustained reduction in blood pressure.^[36] Oplander *et al.*^[33] found that total body irradiation with UVA radiation, acutely induced a significant increase in flow-mediated vasodilation and a transient fall in mean arterial pressure associated with an increased release of nitric oxide from cutaneous photo labile nitric oxide (NO) derivatives. Hence, exposure of human skin to physiologically relevant quantities of UVA component of sunlight leads to a fall in blood pressure, independently of temperature change.

Other mechanism for cardiovascular protection is through vitamin D photosynthesis by sunlight. Vitamin D, the sunshine hormone is formed by ultraviolet B (UVB)-mediated photolysis of 7-dehydrocholesterol in the skin.^[37] A study has reported that an increased 25(OH)D concentration is associated with reduced risk of hypertension^[38], but oral vitamin D supplementation has no effect on BP and cardiovascular morbidity or mortality.^[39] In 1998, Krause *R et al.* reported that short term ultraviolet B exposure had blood pressure lowering effect in patients with untreated mild hypertension with increase of plasma 25(OH)D concentrations.^[40] Vitamin D deficiency is associated with suppression of resting vagal tone, and this results in suppression of the sympathovagal balance, following a withdrawal of the cardio-protective vagal tone.^[41] Observational studies have also shown that higher vitamin D status will improve lipid levels^[42], which will also have beneficial role in reducing cardiovascular disease risk. The prevalence of hypertension and mean diastolic and systolic blood pressures correlates directly with latitude and altitude, being higher in populations living afar from the equator.^[43, 44] Reduced epidermal vitamin D3 photosynthesis due to decreased UV light intensity at distances from the equator and along with increased parathyroid hormone secretion which in turn stimulates the growth of vascular smooth muscles and enhances its

contractility by affecting intracellular calcium, adrenergic responsiveness, and/or endothelial function.^[45,46] By improving vitamin D3 level through sunshine, it will be possible to inverse the parathyroid hormone levels^[47] and hence to normalize the calcium homeostasis and thereby it would be possible to reduce the blood pressure. Thus, improvement of vitamin D status by serial appropriate doses of ultraviolet radiation by sunlight can reduce the risk, and can increase the health related quality of life status in patients with hypertension.

The other mechanism that would be involved in vascular protective action of sunlight is through the production of substance P. Substance P is a potent dilator of many vascular beds.^[48] When skin is exposed to the sunlight, it has the potential to produce substance P.^[49] Substance P is involved in the axon reflex-mediated vasodilatation to local heating. Local heat production will also be encountered during sun exposure. And this mechanism would also be involved in blood pressure reduction following sunbath.

Significant seasonal variation in plasma levels of cholesterol with higher values in winter has been reported in many studies^[50,51], and this has influence on blood pressure. Serum cholesterol is strongly associated with endothelial dysfunction and reduced nitric oxide bioavailability, which will lead to functional arterial stiffening.^[52] Hence controlled exposure to sunlight will have beneficial effects on serum cholesterol levels and improves bioavailability of nitric oxide and there by provides cardiovascular protection. Sunlight exposure will also have potential to produce serotonin^[53] that will help in improving the feel good experience of individual thereby would involve in the pathway of reducing elevated blood pressure.

Being natural reward mechanism encouraging sun exposure, β -endorphins^[54] will induce a feeling of wellbeing, reduces the stress and helps in maintaining homeostasis thereby would help in reverting the blood pressure to safer range.

Physical activity positively affects BP by improving the endothelial functioning.^[55] Exercise increases blood flow leading to increased shear stress, and those results in endothelium-dependent, flow-mediated dilation of vessels. Chronic increase in shear stress will help to improve endothelial function. Physical activity will also reduce the elevated sympathetic nerve activity.^[56] Levels of physical activity are most often found higher in summer than in winter both in men and women.^[57]

Epidemiological studies have reported that systolic and diastolic blood pressure decreases in association with increasing outdoor temperature.^[58] Cold weather will exert a body cooling effect, resulting in increased stress on the heart, increasing blood coagulability, and will increase the risk of developing CVD. And also cold

weather will cause personal changes in lifestyle such as dietary changes, increased weight, or decreased physical activity with consequent increased risk of venous thrombus formation, which increases the risk of developing CVD. People living in the colder climates may also develop hypertension which is related to the polymorphisms in genes controlling adaptation to cold temperature of the environment.^[59] And hence, ambient insolation will definitely have higher temperature at the outdoor places and on a long run that would help in lowering blood pressure.

Hence it can be suggested that sunbath has cardiovascular protective action by increasing the parasympathetic activity with a simultaneous sympathetic withdrawal. By the production of potent vasodilators and through various pathways which are headed mainly by significant production of vitamin D in the cutaneous surface as the beneficial mechanisms underlying the positive effect of sunbath in hypertensive individuals. Even small UV-mediated reductions in blood pressure will definitely have its positive impact on the burden of disease and moderate exposure to sunlight will also reduce the economic burden caused by hypertension. Hence this study adds to the body of literature which shows exposure to ambient sunshine will have cardiovascular protective actions.

Limitations of the study

- Blinding of the subjects were not done during the study
- Prolonged assessments were not done
- Effects of sunbath were noted according to the data collected only for a day

Directions for future research

The study can be conducted with larger sample size and prolonged monitoring of the outcomes can be done. This can be incorporated along with the other antihypertensive treatment regimens and possible underlying mechanisms can also be studied in depth. Further benefits of sunlight exposure could be studied and applied to treat other non-communicable comorbidities accompanying hypertension.

CONCLUSION

By observing the results from all measured parameters; it can be concluded that, sunbath enhances the parasympathetic activity. It is a better method to modulate the activity of autonomic nervous system by improving parasympathetic activity and it can be applied in clinical practices mainly for reducing the systolic and diastolic blood pressures. Even small UV-mediated reductions in blood pressure will definitely have its positive impact on the burden of disease and moderate exposure to sunlight will also reduce the economic burden caused by hypertension. Hence this study adds to the body of literature which shows exposure to ambient sunshine will have cardiovascular protective actions. And sunbath is also applicable for the disease prevention

and health promotion. Finally, the results of the present study justifies that, exposure to ambient sunshine as a part of our daily routine will help in positive promotion of health by preventing the causation disease and also helps to improve the quality of life.

Abbreviations

CAM: Complementary and alternative, CVD: Cardiovascular disease, DALYs: Disability adjusted life years, DBP: Diastolic Blood pressure, HF: High frequency, HR: Heart rate, HRV: Heart rate variability, LF: Low frequency, LF/HF: Low frequency/ High frequency Ratio, MAP: Mean Arterial Pressure, Mean-RR: Mean of R-R interval, NN50: Normal sinus to normal sinus intervals, NO: Nitric oxide, RMSSD: Root mean square of the successive differences, SBP: Systolic Blood pressure, UVR: Ultraviolet radiation, VLF: Very low frequency, WHO: World health organization, 25(OH)D: 25 hydroxyvitamin D.

Conflict Of Interest

The authors declare that they have no conflict of interest.

ACKNOWLEDGEMENT

We would like to thank SDM management & the Principal for providing all the facilities for conducting the research work.

REFERENCES

1. Mohan V, Deepa R. Risk factors for coronary artery disease in Indians. *The J Assoc Phys India*, 2004 Feb 1; 52: 95-7.
2. Kaplan NM. Systemic hypertension: mechanisms and diagnosis. *Heart disease*, 1997; 235-47.
3. Beevers G, Lip GY, O'Brien E. The pathophysiology of hypertension. *Bmj*, 2001 Apr 14; 322(7291): 912-6.
4. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, Roccella EJ, Stout R, Vallbona C, Winston MC, Karimbakas J. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. *Jama*, 2002 Oct 16; 288(15): 1882-8.
5. Clark LT. Improving compliance and increasing control of hypertension: needs of special hypertensive populations. *Am. Heart J.*, 1991 Feb 1; 121(2): 664-9.
6. Tabish SA. Complementary and alternative healthcare: is it evidence-based?. *IJHS*, 2008 Jan; 2(1): V.
7. Tada T. Toward the philosophy of CAM: super-system and epimedical sciences. *Evidence-based Complementary and Alternative Medicine*, 2004; 1(1): 5-8.
8. Oberg EB, Bradley R, Cooley K, Fritz H, Goldenberg JZ, Seely D, Saxton JD, Calabrese C. Estimated effects of whole-system naturopathic medicine in select chronic disease conditions: A

- systematic review. *Alternative & Integrative Medicine*, 2015 Apr 24.
9. Nair P, Nanda A. Naturopathic medicine in India. *Focus on Alternative and Complementary Therapies*, 2014; 19(3): 140-147.
 10. Lubna, Sherani FS. Heliotherapy: then and now - a review. *IJRAR*, 2018; 5(4): 657-662.
 11. Baaghdeh M, Mayvaneh F. Climate change and simulation of cardiovascular disease mortality: A case study of Mashhad, Iran. *Iran. J. Public Health*, 2017 Mar; 46(3): 396.
 12. De Blois J, Kjellstrom T, Agewall S, Ezekowitz JA, Armstrong PW, Atar D. The effects of climate change on cardiac health. *Cardiology*, 2015; 131(4): 209-17.
 13. Rostand SG. Ultraviolet light may contribute to geographic and racial blood pressure differences. *Hypertension*, 1997 Aug; 30(2): 150-6.
 14. Rostand SG, McClure LA, Kent ST, Judd SE, Gutiérrez OM. Associations of Blood Pressure, Sunlight, and Vitamin D in Community-Dwelling Adults: The Reasons for Geographic and Racial Differences in Stroke (Regards) Study. *J. of Hypertens*, 2016 Sep; 34(9): 1704.
 15. Liu D, Fernandez BO, Hamilton A, Lang NN, Gallagher JM, Newby DE, Feelisch M, Weller RB. UVA irradiation of human skin vasodilates arterial vasculature and lowers blood pressure independently of nitric oxide synthase. *J. Investig. Dermatol*, 2014 Jul 1; 134(7): 1839-46.
 16. Patil S S, Taklikar R H. A Comparative Study of Heart Rate Variability During Deep Breathing in Normotensive and Hypertensive Subjects. *IJMRHS*, 2015; 4(4): 421-425.
 17. Yildirim A, Kabakci G, Akgul E, Tokgozoglu L, Oto A. The effects of menstrual cycle on cardiac autonomic innervation as assessed by heart rate variability. *J Am Coll Cardiol*, 2002; 39: 208.
 18. Takahashi H, Yoshika M, Yokoi T. Validation of three automatic devices for the self-measurement of blood pressure according to the European Society of Hypertension International Protocol revision 2010: the Omron HEM-7130, HEM-7320F, and HEM-7500F. *Blood pressure monitoring*, 2015 Apr 1; 20(2): 92-7.
 19. Telles S, Nagarathna R, Nagendra H. Physiological Measures of Right Nostril Breathing. *J Altern Complem Med*, 1996; 2(4): 479-484.
 20. Fulbrook P: Core temperature measurement in adults: a literature review. *J Adv Nurs*, 1993; 18: 1451-60.
 21. Babbitt ED. *Human culture and cure*. Hyderabad: Prakritiprakashan, 1972.
 22. Tarveinen MP, Niskanen JP, Lipponen JA, Rantaho PO, Karjalainen PA. Kubios HRV – Heart rate variability analysis software. *Computer methods and programs in biomedicine*, 2014; 113(1): 210-220.
 23. Elizabeth H. Sun's U Light Helped Spa k Life. *Origin And Evolution Of Life*, 2017 Mar 30.
 24. Krause R, Stange R, Kaase H, Holick MF. UV irradiation and pleiotropic effects of vitamin D in chronic kidney disease—Benefits on cardiovascular comorbidities and quality of life. *Anticancer research*, 2016 Mar 1; 36(3): 1403-8.
 25. Lehmann P, Hölzle E, Melnik B, Plewig G. Effects of ultraviolet A and B on the skin barrier: a functional, electron microscopic and lipid biochemical study. *Photodermatology, photoimmunology & photomedicine*, 1991 Jun; 8(3): 129-34.
 26. Chowdhury R, Kunutsor S, Vitezova A, Oliver-Williams C, Chowdhury S, Kieft-de-Jong JC, Khan H, Baena CP, Prabhakaran D, Hoshen MB, Feldman BS. Vitamin D and risk of cause specific death: systematic review and meta-analysis of observational cohort and randomised intervention studies. *Bmj*, 2014 Apr 1; 348: g1903.
 27. Lucas RM, McMichael AJ, Armstrong BK, Smith WT. Estimating the global disease burden due to ultraviolet radiation exposure. *Int J Epidemiol*, 2008 Jun 1; 37(3): 654-67.
 28. Goyal A, Aslam N, Kaur S, Soni RK, Midha V, Chaudhary A, Dhaliwal LK, Singh B, Chhabra ST, Mohan B, Anand IS. Factors affecting seasonal changes in blood pressure in North India: a population based four-seasons study. *Indian Heart J.*, 2018 May 1; 70(3): 360-7.
 29. Sinha P, Taneja DK, Singh NP, Saha R. Seasonal variation in prevalence of hypertension: Implications for interpretation. *Indian J. Public Health*, 2010 Jan 1; 54(1): 7.
 30. Weller RB, Wang Y, He J, Maddux FW, Usvyat L, Zhang H, Feelisch M, Kotanko P. Does Incident Solar Ultraviolet Radiation Lower Blood Pressure. *J Am Heart Assoc*, 2020 Mar 3; 9(5): e013837.
 31. Scragg R, Rahman J, Thornley S. Association of sun and UV exposure with blood pressure and cardiovascular disease: A systematic review. *J. Steroid Biochem. Mol. Biol.*, 2019 Mar 1; 187: 68-75.
 32. Moncada S, Higgs EA. The discovery of nitric oxide and its role in vascular biology. *Br. J. Pharmacol*, 2006 Jan; 147(S1): S193-201.
 33. Opländer C, Volkmar CM, Paunel-Görgülü A, van Faassen EE, Heiss C, Kelm M, Halmer D, Mürtz M, Pallua N, Suschek CV. Whole body UVA irradiation lowers systemic blood pressure by release of nitric oxide from intracutaneous photolabile nitric oxide derivatives. *Circulation research*, 2009 Nov 6; 105(10): 1031-40.
 34. Feelisch M, Kolb-Bachofen V, Liu D, Lundberg JO, Revelo LP, Suschek CV, Weller RB. Is sunlight good for our heart. *Eur Heart J*. 2010 May 1; 31(9): 1041-5.
 35. Dejam A, Kleinbongard P, Rassaf T, Hamada S, Gharini P, Rodriguez J, Feelisch M, Kelm M. Thiols enhance NO formation from nitrate photolysis. *Free Radical Biology and Medicine*, 2003 Dec 15; 35(12): 1551-9.

36. Suschek CV, Opländer C, van Faassen EE. Non-enzymatic NO production in human skin: effect of UVA on cutaneous NO stores. *Nitric Oxide*, 2010 Feb 15; 22(2): 120-35.
37. Wacker M, Holick MF. Sunlight and Vitamin D: A global perspective for health. *Dermato-endocrinology*, 2013 Jan 1; 5(1): 51-108.
38. Judd S, Tangpricha V. Vitamin D deficiency and risk for cardiovascular disease. *Circulation*, 2008 Jan 29; 117(4): 503.
39. Pilz S, Gaksch M, Kienreich K, Grübler M, Verheyen N, Fahrleitner-Pammer A, Treiber G, Drechsler C, ó Hartaigh B, Obermayer-Pietsch B, Schwetz V. Effects of vitamin D on blood pressure and cardiovascular risk factors: a randomized controlled trial. *Hypertension*, 2015 Jun; 65(6): 1195-201.
40. Krause R, Bühring M, Hopfenmüller W, Holick MF, Sharma AM. Ultraviolet B and blood pressure. *Lancet (British edition)*, 1998; 352(9129): 709-10.
41. Mann MC, Exner DV, Hemmelgarn BR, Sola DY, Turin TC, Ellis L, Ahmed SB. Vitamin D levels are associated with cardiac autonomic activity in healthy humans. *Nutrients*, 2013 Jun; 5(6): 2114-27.
42. Jorde R, Grimnes G. Vitamin D and metabolic health with special reference to the effect of vitamin D on serum lipids. *Progress in lipid research*, 2011 Oct 1; 50(4): 303-12.
43. Mingji C, Onakpoya IJ, Perera R, Ward AM, Heneghan CJ. Relationship between altitude and the prevalence of hypertension in Tibet: a systematic review. *Heart*, 2015 Jul 1; 101(13): 1054-60.
44. Rostand SG. Ultraviolet light may contribute to geographic and racial blood pressure differences. *Hypertension*, 1997 Aug; 30(2): 150-6.
45. Bian K, Ishibashi KA, Bukoski RD. 1, 25 (OH) 2D3 modulates intracellular Ca²⁺ and force generation in resistance arteries. *Am J Physiol Heart Circ Physiol*, 1996 Jan 1; 270(1): H230-7.
46. Schleiffer R, Pernot F, Jones R. Endothelium is a target organ of parathyroid secretions in genetic hypertensive rats. *Hormone and metabolic research*, 1995 Jan; 27(01): 16-8.
47. Martins JS, Palhares MD, Teixeira OC, Gontijo Ramos M. Vitamin D status and its association with parathyroid hormone concentration in Brazilians. *Int. J. Nutr. Metab.*, 2017; 2017.
48. Watson RE, Supowit SC, Zhao H, Katki KA, Dipette DJ. Role of sensory nervous system vasoactive peptides in hypertension. *Braz J Med Biol Res.*, 2002 Sep; 35(9): 1033-45.
49. Mead MN. Benefits of sunlight: a bright spot for human health, 2008 Apr; 116(4): A160-A167.
50. Fyfe T, Dunnigan MG, Hamilton E, Rae RJ. Seasonal variation in serum lipids, and incidence and mortality of ischaemic heart disease. *J. Atheroscler. Res.*, 1968 Jan 1; 8(3): 591-6.
51. Gordon DJ, Trost DC, Hyde JO, Whaley FS, Hannan PJ, Jacobs Jr DR, Ekelund LG. Seasonal cholesterol cycles: the Lipid Research Clinics Coronary Primary Prevention Trial placebo group. *Circulation*, 1987 Dec; 76(6): 1224-31.
52. Wilkinson IB, Cockcroft JR. Cholesterol, endothelial function and cardiovascular disease. *Current opinion in lipidology*, 1998 Jun 1; 9(3): 237-42.
53. Slominski A, Wortsman J, Tobin DJ. The cutaneous serotonergic/melatonergic system: securing a place under the sun. *The FASEB Journal*, 2005 Feb; 19(2): 176-94.
54. Fell GL, Roinson KC, Mao J, Woolf CJ, Fische DE. Skin β -endorphin mediates addiction to UV light. *Cell.*, 2014 Jun 19; 157(7): 1527-34.
55. Wareham NJ, Wong MY, Hennings S, Mitchell J, Rennie K, Cruickshank K, Day NE. Quantifying the association between habitual energy expenditure and blood pressure. *Int J Epidemiol*, 2000 Aug 1; 29(4): 655-60.
56. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. Exercise and hypertension. *Medicine & Science in Sports & Exercise*, 2004 Mar 1; 36(3): 533-53.
57. Dannenberg AL, Keller JB, Wilson PW, CASTELLI WP. Leisure time physical activity in the Framingham Offspring Study: description, seasonal variation, and risk factor correlates. *Am. J. Epidemiol*, 1989 Jan 1; 129(1): 76-88.
58. Madsen C, Nafstad P. Associations between environmental exposure and blood pressure among participants in the Oslo Health Study (HUBRO). *Eur. J. Epidemiol*, 2006 Jul 1; 21(7): 485-91.
59. Leow MK. Environmental origins of hypertension: phylogeny, ontogeny and epigenetics. *Hypertension Research*, 2015 May; 38(5): 299-307.