Publisher
Maharshi Charaka Ayurveda
www.maharshicharaka.in

E-ISSN:2456-3110

Journal of Ayurveda and Integrated Medical Sciences

2021 Volume 6 Number 1 Jan-Feb

Research Article
Clinical Trial

Effect of Integrated Approach of Yoga Therapy (IAYT) on DNA damage

Ghimire A.[1*], Ghimire B.[2], Neupane S.[3], Pokharel S.[4], Pokharel S.[5]

DOI: 10.21760/jaims.6.1.6

[1*] Amrita Ghimire, PhD Scholar, Nepal Sanskrit University, Dang, Nepal.

[2] Bikash Raj Ghimire, Senior Consultant, National Ayurveda Research and Training Centre, Kathmandu, Nepal.

[3] Samichha Neupane, Consultant, District Ayurveda Health Centre, Kavre, Nepal.

[4] Sanjay Pokharel, Professor, Nepal Sanskrit University, Dang, Nepal.

[5] Shwetha Kumari Pokharel, Asso. Professor , Nepal Sanskrit University, Dang, Nepal.

Aim: To assess the effect of IAYT on extent of betterment of DNA damage in diabetic practicing Yoga. **Method:** Thirty participants with diabetes recruited from Arogyadhama, Holistic Health Care Home in PrashantiKutiram, at Bangalore, were checked for DNA damage before and after 7 days of IAYT intervention. Age range of participants was from 30 to 65 years.Intervention consisted of intensive residential Yoga program comprising of Asana (physical posture), Pranayama, meditation, devotional sessions, diet modification and interactive sessions on philosophical concepts of Yoga. The damage in the genomic DNA of peripheral blood mononuclear cells was assayed by single cell gel electrophoresis method following the previously described protocol of Singh et al. Analysis of the data was done using CASP and excel. **Result:** After one week of IAYT program, 63% of participants showed lessening in the DNA damage (decrease in post tail length) after intervention while in 37% the DNA damage increased (increase in post tail length). The percentage of decrease of tail length was significantly higher (37%, comparing the percentage of means of pre and post) than the percentage of increase of tail length (19%). The data of both positive and negative change showed normal distribution. **Conclusion:** DNA Damage has a direct link with defects in metabolism, non-communicable disease like diabetes andstress. There was significant reduction in the tail length of DNA and the total comet length after IAYT intervention which signifies a better improvement in the DNA damage. Whether this was achieved because of reduction in stress or through another physiological pathway is not known. Normal distribution of data shows that the damage or betterment of damage, are both not a chance occurring in this data but first of a kind report of Yoga influencing the DNA repair mechanism. Also, in this novel study we have made findings to differentiate between short and long fragment DNA damage through data analysis.

Keywords: DNA damage, Electrophoresis, Cometlength, DNA repair, diabetes, stress, IAYT

Corresponding Author

Amrita Ghimire, PhD Scholar, Nepal Sanskrit University, Dang, Nepal.
Email: meetkusu@gmail.com

How to Cite this Article

Amrita Ghimire, Bikash Raj Ghimire, Samichha Neupane, Sanjay Pokharel, Shwetha Kumari Pokharel, Effect of Integrated Approach of Yoga Therapy (IAYT) on DNA damage. J Ayu Int Med Sci. 2021;6(1):39-48.
Available From https://www.jaims.in/index.php/jaims/article/view/1179

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Manuscript Received	Review Round 1	Review Round 2	Review Round 3	Accepted
2021-01-12	2021-01-21	2021-01-26	2021-02-03	2021-02-06

Conflict of Interest	Funding	Ethical Approval	Plagiarism X-checker	Note
Nil	Nil	Yes	9%	

Introduction

DNA or deoxyribonucleic acid, present inside the nuclear membrane in all living creatures act as a repository of information for the functioning of the cell and also carries information from one generation to another. The two strands are held together by hydrogen bonds (Vella, 1994). Each strand is a long chain of nucleotides and each nucleotide subunit is composed of three components: deoxyribose sugar, a phosphate group, and nitrogenous base. In the pentose sugar the 2 carbon atoms at position 5 and 3 forms link with a phosphate and a hydroxyl group respectively. The phosphate group of one nucleotide forms bond with a hydroxyl group of the subsequent nucleotide through phosphodiester bonds. This alternating sugar and phosphate forms the backbone of the DNA strands. On the 2 strands of DNA helix the backbone run in anti-parallel direction, one strand runs in the 5′ to 3′ while the other runs in 3′ to 5′ direction, (Sinden, 1961). The information in DNA is stored as a code made up of four chemical bases. They are adenine (A), guanine (G), cytosine (C), and thymine (T). Adenine and guanine together are called purines. Thymine and cytosine are called pyrimidine. The number of purine bases is equals and opposite to the number of pyrimidine bases. Adenines and thymine are held by two bond and guanine and cytosine are held by three bond (Vella, 1994). The double helix of DNA forms a narrow thread like structure with a diameter of 2nm and a total length of 2 m and it could conceivably be compacted into a tight ball like a ball of string (Vella, 1994), this compacted form of DNA is known as chromosome. DNA consists of about 3 billion bases, and more than 99 percent of those bases are the same in all people (Franklin, Crick, & Mendel, 2007).

DNA Damage

DNA damage is caused due to continuous exposure of the genome to various endogenous and environmental agents producing a variety of DNA lesions. These lesions can affect the fidelity of DNA replication (Painter, 1985), and transcription which can create mutations. As a result, the produced mutated protein can affect various biological processes leading to the genome instability (Protic-Sabljic and Kraemer, 1985). If these damages occur in the germ cells, it can be hereditary and will be harmful to the next generation in passing a genetic disease. The DNA damage can have genotoxic and cytotoxic effects on the cell (Tuteja, Singh, Misra, Bhalla, &Tuteja, 2001). DNA damage has been shown to either promotes anti-cancer, anti-aging response or enhance development of premature aging pathologies including obesity, hypertension, diabetes, show disintegration of DNA repair mechanisms, resulting in accumulation of damaged DNA. With continuous accumulation of DNA damage, the body resorts to maintenance mode, where the growth will cease which is seen in premature aging pathologies and senescence (Li, 2008).

The other triggering factors are mutagens, radiation and even inevitable replication process of DNA. DNA damage from all endogenous sources give rise to some 20,000 lesions per day per cell. Calculation considering all possible processes causing DNA damage reports that there can be as much as 3 x 10_{17} DNA damaging events per hour in a human body. Strictly, DNA molecule can be said to be damaged when there is any abnormality from regular double stranded structure, (Drabløs et al., 2015). DNA damages include covalent changes in DNA structures and noncovalent anomalous structures, including base pair mismatches, loops, and bubbles arising from a string of mismatches (Li, 2008). Change in structure can happen because of one of the following, (1) single base change, (2) structural distortion or (3) DNA backbone damage. Single base changes occur due to deamination, alkylation or oxidation of the bases resulting in the formation of transversion of bases from GC to AT. Most common structural alteration is caused by the cross-linked Thymine dimmers while DNA backbone damage, which is the most severe type of DNA damage, is seen when there are double strand breaks.

Stability of the genome is supported by an intricate machinery of repair, damage tolerance and checkpoint pathways that counteracts DNA damage. Response of the cell to DNA damage can be, bypassing it, reversing it or replacing the damaged part. Bypassing mechanism is taken care mostly by different kinds of DNA polymerase and the reversal is done by methyltransferases. In damage removal process of managing DNA damage there are four mechanisms, base excision repair, mismatch repair, nucleotide excision repair and double-strand break repair involving a wide variety of proteins is a well-established link between disintegration of DNA repair mechanisms.

Stress

Stress can be studied under both Psychological and physiological domains both of which are very tightly interlinked. Stress can be characterized as a negative emotional experience accompanied by predictable biochemical, physiological, cognitive, and behavioral changes that are directed either towards altering the stressful event or accepting to its effect. The initial response of the body to stressful situations may be arousal, excessive reactivity, and enervation, leading to cumulative damage to the body system (Taylor, 2008). Continues exposure to stress can lead to physiological and psychological symptoms such as anxiety and depression (Arora, S, 2008).

DNA damage due to psychological stress

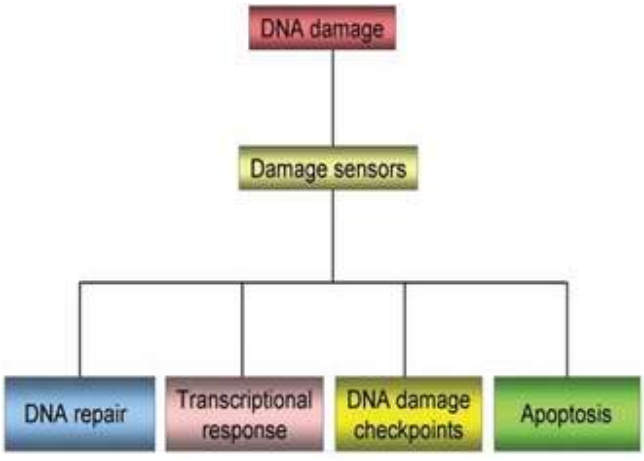
Psychological stress arises when an individual perceives that environmental demands exceed above his or her adaptive capacity (Cohen, Janicki-Deverts, & Miller, 2007). DNA damage is increased due to the constant exposure with stress and stress hormones (cortisol and catecholamine). It is suggested that chronic stress is through β-adrenergic stimulation which can induce two synergistic molecular pathways that result in build-up of DNA damage. Psychological stress has been shown to activate the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system, resulting increases in cortisol and catecholamine. The effects of catecholamine are facilitated by nine distinct α-adrenergic and β-adrenergic G-protein-coupled receptors which are present inside the cell (Flint &Bovbjerg, 2012). Immobilization stress starts damage at cellular, and at the level of molecules level like protein, lipid and DNA which is the main causes of disease and aging such as brain dysfunctions and immune system degeneration (Liu et al., 1996).

DNA damage due to oxidative stress

Cells in tissues and organs are continuously exposed to oxidative stress and free radicals on a daily basis. These free radicals are generated during endogenous (intracellular) metabolism or by exogenous agent. Primary exogenous factor are radiation, cosmic rays and alpha particle and endogenous factor are cellular signaling and metabolic processes. DNA lesion induced endogenously is much higher than the one persuaded exogenously (Kryston, Georgiev, Pissis, & Georgakilas, 2011). DNA damage is caused due To oxidative stress which effect a series of metabolic event within the cells that leads to activation of nuclease enzymes which damage the backbone of DNA (Halliwell & Aruoma, 1991). Oxidative stressresults when reactive oxygen species are not adequately removed. This can happen if antioxidants are depleted or if the formation of reactive oxygen species is increased beyond the ability of the defenses to cope with them which can result in severe metabolic dysfunctions, including peroxidation of membrane lipids, depletion of nicotinamide nucleotides, rises in intracellular free Calcium ions, cytoskeleton disorder and DNA damage (Halliwell & Aruoma, 1991).

Commonly produced free radicals are superoxide (O), nitric oxide (NO+) and OH radicals. These are not highly reactive but under certain conditions it generates toxic and cause irreversible/reversible damage in nucleic acid, amino acid, lipid, protein, lipoprotein, carbohydrates and connective tissues. This types of free radical are formed due to inflammation, aging, induced by exercise, cancer and many other traumatic disease (Michalsen, A et al., 2012).

Figure 1: DNA damage response reactions in mammalian cells (Sancar& Lindsey-boltz, 2004).



Aims and Objective

Aim

To assess the Effect of IAYT on DNA damage.

Objectives

01. To assess the extent of betterment of DNA damage in people practicing Yoga.
02. To evaluate the effect of Yoga in addressing DNA damage due to stress

Materials and Methods

Subjects: People with T2DM.

Source of subjects: Subjects were recruited from Arogyadhama, Holistic Health Care Home in Prashantikutiram.

Age: 30-65 years.

Gender: Both Male & Female.

Sample Size: n= 30.

Inclusion Criteria

- People with T2DM.
- Between ages range of 35-55 years.
- Both male and female subjects willing to participate.
- Arogyadhama participants practicing IAYT module for diabetes

Exclusion Criteria

- Subjects who are bedridden
- Attendance less than 70% to the intervention sessions

Informed Consent: A signed informed consent was obtained from all the subjects after explaining about the trial.

Collection of Blood Samples: Blood samples (3 ml) of the subjects were collected and stored in EDTA tubes (30µl) and were transported to the laboratory for further test.

Hypothesis

Effect of IAYT may improve the DNA repair mechanisms.

Design: Single pre-post design.

Intervention

Integrated Approach of Yoga Therapy

All participants will practice *Asanas*twice a day (each 60min), *Pranayama*& OM meditation (60min), cyclic meditation (50min), MSRT (30min), *Trataka* (30min), *Bhajan* (30min), happy assembly (60min) and lecture (60min) for the period of one week.

Twice of kriya sessions will be given in a week.

Table 1: Yoga based lifestyle routine under IAYT intervention

Time	Schedule
05:30 am	Pranayama, Om meditation
06:30 am	Yogasanas session 1 (tailor made based on individual complaints)
07:30 am	Breakfast
08:00 am	Lecture on lifestyle advises as per Bhagawad Gita
09:00 am	Consultation with Integrative Medicine Physicians
09:45 am	Yogic Counseling
12:15 pm	Lecture on Stress Management: yogic and modern concepts
01:00 pm	Lunch
02:00pm	Karma Yoga
03:00pm	Cyclic Meditation
04:00 pm	Yogasanas session 1 (tailor made based on individual complaints)
05:00 pm	Walk and tuning to nature
06:00 pm	Bhajan (devotional sessions)
06:30 pm	Trataka, MSRT (advanced meditations)
07:30 pm	Dinner

Assessments

The assessment was done before and after intervention. The only assessment done in this study was to assess the DNA damage assessed through comet assay also called single cell gel electrophoresis (SCGE), developed by N.P. Singh, combines the simplicity of biochemical techniques for detecting DNA single strand breaks (frank strand breaks and incomplete excision repair sites), alkali-labile sites and crosslinking with the single cell approach typical of cytogenetic assays. Following is the protocol in brief as described by N.P. Singh (Dhawan et al., 2003).

Protocol in brief

01. Slides are prepared by coating 1% Normal Melting Agarose (NMA) solution.
02. To the coated slide, add 75 µL of Low Melting Point Agarose (LMPA) (0.5%; 37ºC) mixed with ~10,000 lymphocytes in ~5-10 µL of whole blood and a third layer of LMPA is poured and allowed to solidify by placing at 4o
03. After about 2 hrs in alkaline buffer (pH >13) place the slides in the electrophoresis tank and the electrophoresis done 24V and 300mA for 30 mins.
04. After electrophoresis gently lift the slide and stain with 1% ethidium bromide for 5 mins.

05. Drain the slides and dehydrate by immersing in cold 100% ethanol and dry at 50oC in incubator.
06. Rehydrate the slides before imaging with cold water for 30 mins and stain again with 1% ethidium bromide.
07. Visualize in gel documentation system with CCD camera to assess the quantitative and qualitative extent of DNA damage in the cells by measuring the length of DNA migration and the percentage of migrated DNA. Generally, 50 to 100 randomly selected cells are analyzed per sample.

Outcome measure

The outcome measure of this exploratory study is to provide us insights about the DNA repair mechanisms following Yoga practices in chronic medical illness. Yoga is widely accepted as a reliever of stress which it accomplishes by enhancing the parasympathetic tone which is required for managing many of the common NCDs. Elucidation of scientific evidence for such benefits accrued from Yoga is the one of the primary outcomes. This study with more detailed analysis of the results should also provide clues to the pathways (of DNA repair) which Yoga practices targets to help in combating various disease conditions.

Data analysis

The data were analysed using the software CASP which measure the tail length and comet length of DNA based on the pixel intensities. The pixel intensities measure the length of the comet tail, percentage of DNA in the head and tail of the comet and the total comet length, which is the total of tail and head length of the comet. In each sample a minimum of 30 comets were analysed from more than one slide. The details were taken as tab delimited columns into an excel file for further analysis.

Results

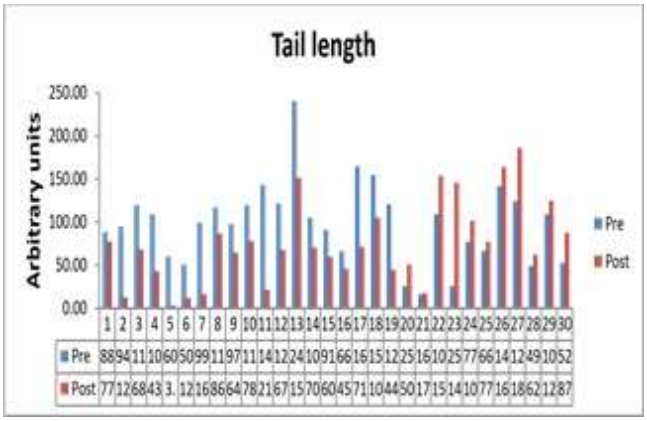
A single group pre-post design was conducted to study the efficacy of Integrated Approach of Yoga Therapy in people having diabetes. The objective of the analysis was to find out the differences in the tail length of the comet produced as a result of electrophoresis. The parameters of tail length, comet length, Head DNA percent (Head DNA%) and Tail DNA percent (tail DNA %) measured, as described in the preceding sections, from 30 comets were averaged (Table 2). Most of the comparisons Between pre & post were done using these averages. The tail length (used as a measure of DNA damage) was seen both increasing and decreasing, signifying that the extent of DNA damage both increased and decreased respectively. In 19 out of 30 participants the DNA damage decreased while it increased in the remaining 11 participants (Figure 2). Also, the data showed the normal distribution pattern for all the parameters measured, one for tail length is shown below (Figure 3).

Table 2: Means of pre & post intervention

Pre					Post			
Sample No.	HeadDNA%	TailDNA %	TailLength	CometLength	HeadDNA%	TailDNA%	TailLength	CometLength
Sample 2	45.6316	54.3684	87.3913	119.4347826	48.81118	51.18882	77.4	114.48
Sample 3	54.24744	45.75255	94.88	129.8	94.45209	5.547911	12.83333	59.58333
Sample 4	38.83599	61.16401	119.8	154.48	63.75863	36.24137	68.29167	102.9583
Sample 5	37.65441	62.34559	109.12	139.8	76.38648	23.61352	43.20833	87.125
Sample 20	58.88314	41.11686	60.04	95.36	97.75968	2.240313	3.44	22.6
Sample 23	63.15776	36.84224	50.72	79.16	91.7849	8.215109	12.16	52.28
Sample 26	41.99192	58.00808	99.28	126.68	90.27704	9.722963	16.2	61.44
Sample 28	60.56066	39.43934	117.12	155.4	56.11749	43.88251	86.68	132.96
Sample 29	52.69261	47.30739	97.68	133.56	77.27334	22.72664	64.68	108.24
Sample 30	48.3864	51.6136	119.48	163.12	64.10008	35.89991	78.04	122.48
Sample 33	38.70118	61.29882	143.28	176.2	82.21648	17.78352	21.4	48.08
Sample 36	51.76042	48.23958	121.6	167	64.0225	35.9775	67.84	105
Sample 61	35.78003	64.21997	240.48	300.04	56.59322	43.40678	151.24	222.96
Sample 63	63.48189	36.51811	105.32	171.28	64.51594	35.48406	70.52	126.4
Sample 66	34.94247	65.05753	91	127.68	44.74715	55.25285	60	95
Sample 67	39.46713	60.53287	66.36	97.6	62.15437	37.8456	45.92	85.64
Sample 68	31.58134	68.41866	164.92	197.6	57.56156	42.43843	71.36	106.76
Sample 69	35.76183	64.23818	154.8	197.4	53.75561	46.24439	105.2	153.16

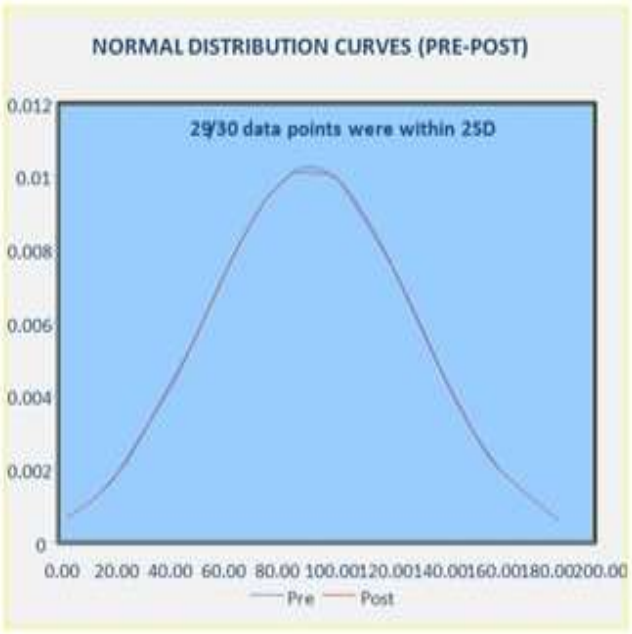
Sample36	51.76042	48.23958	121.6	167	64.0225	35.9775	67.84	105
Sample61	35.78003	64.21997	240.48	300.04	56.59322	43.40678	151.24	222.96
Sample63	63.48189	36.51811	105.32	171.28	64.51594	35.48406	70.52	126.4
Sample66	34.94247	65.05753	91	127.68	44.74715	55.25285	60	95
Sample67	39.46713	60.53287	66.36	97.6	62.15437	37.8456	45.92	85.64
Sample68	31.58134	68.41866	164.92	197.6	57.56156	42.43843	71.36	106.76
Sample69	35.76183	64.23818	154.8	197.4	53.75561	46.24439	105.2	153.16
Sample70	36.4791	63.5209	120.84	151.84	74.30854	25.69146	44.8	91.4
Sample14	76.08459	23.91541	25.76	60.12	74.17876	25.82122	50.75	89.41667
Sample22	84.53504	15.46496	16.32	42.36	91.1418	8.858186	17.8	54.8
Sample31	55.94186	44.05814	109.08	155.44	36.50957	63.49043	153.92	190.6
Sample41	88.859	11.14101	25.88	64.8	33.50418	66.49582	145.76	180.92
Sample42	71.22384	28.77616	77.28	122.12	48.56059	51.43941	101.56	136.96
Sample50	64.68843	35.31157	66.92	107.68	49.78743	50.21257	77.40909	111.8636
Sample51	61.42657	38.57343	141.9091	213.90	51.69299	48.30701	163.8095	228.0476
Sample52	62.52571	37.47429	124.3333	197.58	56.56364	43.43636	186.04	268.16
Sample62	82.65076	17.34925	49.36	118.6	72.29517	27.70484	62.12	116.32
Sample64	64.96561	35.03439	109.12	179.32	31.61414	68.38586	124.72	153.96
Sample65	75.84314	24.15686	52.72	115.8	54.95363	45.04638	87.56	137.6

Figure 2: Comparison of tail length (pre and post)



The chart shows the pre-post DNA tail length changes. Out of the total sample (n=30) 63% (samples 1-19) have shown decrease in tail length while 37% (samples 20-30) have shown an increase in tail length. Table also shows pre & post tail lengths in arbitrary units.

Figure 3: Normal distribution curve



The extent of DNA damage decreasing was more significant than the increase in damage. Sixty three percent of the participants with diabetes showed 38% decrease in the extent of DNA damage, while 37% participants showed a 19% increase in DNA damage (Table 3 & 4 and Figure 4 & 5).

Table 3: Positive change of tail length

	Mean ± SD	% Change	p-value
Tail length - pre	113.95±42.56	38%	<0.01
Tail length - post	57.96±36.40		

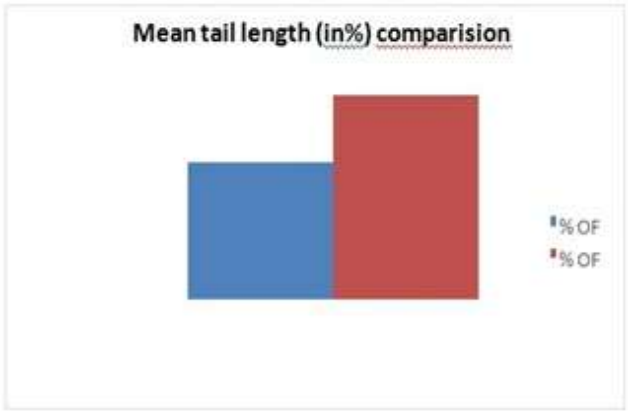
Table is showing a reduction in the tail length after the intervention. The result is from 19 subjects. There was significant reduction in the scores from 113.95±42.56 to 57.96±36.40 with 38 % of change.

Table 4: Negative change of tail length

	Mean ± SD	% Change	p- value
Tail length – pre	72.60±43.21	-19%	<0.01
Tail length – post	106.50±52.83		

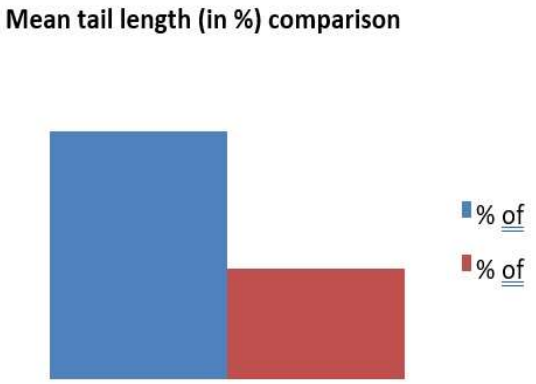
Table showing an increase in the tail length after the intervention. The result is from 11 subjects. There was significant increase in the scores from72.60±43.21 to 106.50±52.83 with -19 % of change.

Figure 4: Negative change of tail length



This bar chart depicts the results of DNA tail length among the subjects after intervention. 19% have shown an increase in the DNA tail length while 38% of the sample has shown an decrease in DNA tail length.

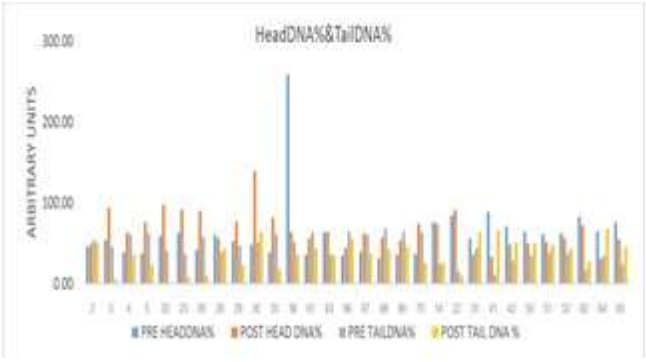
Figure 5: Positive change of tail length



This bar chart depicts the results of DNA tail length among the subjects after intervention. 38% have shown a decrease in the DNA tail length while 19% of the sample has shown an increase in DNA tail length.

The increase or decrease in tail length of the comets was further analysed to see corresponding changes in the head DNA percent and tail DNA percent. The logic was that any increase in the tail length results from DNA moving out of the head into the tail and as a result it can be expected that increase in Tail DNA percent will show a decrease in Head DNA percent (in other words amount of DNA). It was seen that in cases where the DNA damage decreased after intervention (19/30 participants) the Head DNA percent did increase and vice versa in participants who showed increase in DNA damage (11/30 participants) (Figure 6.)

Figure 6: Comparison of percentage of DNA in the head and tail.



This bar chart shows the logic of increase and decrease in tail length. Decrease in tail length after intervention means decrease in tail DNA% and increase in headDNA%. Likewise, increase in tail length after intervention means increase in tailDNA% and decrease in headDNA%.

Discussion

Diabetes mellitus is a complex disease which is associated with many complications, including overweight, atherosclerosis, retinopathy, nephropathy and others. It is known that diabetes is associated with the elevated level of oxidative stress. Stress decreases antioxidative status causing a faster progression of the disease. Formation of oxidants is also a major contributor to aging and the degenerative diseases of aging and damaging cellular molecules (Madhu et al., 2001) including DNA. Yoga-based lifestyle intervention has been shown to be efficacious in reducing oxidative stress and risk of chronic diseases in a short duration. It has been shown to even cause reversal of markers of aging, mainly oxidative stress, telomerase activity, and oxidative DNA damage and also has been shown to decrease the levels of plasma cortisol and interleukin-6 (Kumar, Yadav, Yadav, Tolahunase, & Dada, 2015).

In the current study DNA damage was assessed here using comet assay technique which measures the length of comet and length of individual components of head and tail. Also, the percentage of DNA in head and tail is reported. There was significant reduction in the tail length of DNA ($p<0.01$), total comet length ($p<0.01$) in around 63% of the diabetic participants in whom the DNA damage was analysed pre & post. And the total change percentage also reduced indicating that IAYT intervention helped in a better quality of life and stopping the progression of DNA damage. IAYT

Intervention reduced the tail length of the comet DNA by 38%. Which is this is a very significant change. The probable reason may be adjustment of the lifestyle, removal of stressor from the environment and control of sugar levels in diabetics due to increased physical activity and reduction of stress. Overall, IAYT brings deep relaxation to the system through parasympathetic dominance which is a promoter of sleep. Through yogic counselling deep rooted stressors in sub conscious mind are identified and deeper levels of relaxation are reached. This may drastically reduce the DNA damage. There was also slight increase in the tail length (19%) of the comet in 37% of the participants after intervention. The probable reason may be, participants were unable to cope with the daily schedule of their treatment, sudden change in the environment which increased their stress, they were not able follow the Yoga session properly.

Further analysis of the tail length in relation to percent of DNA in the tail/head revealed that as expected reduced tail length after intervention showed a corresponding increasing in the percent of DNA in the comet head. This is an indication of the betterment of the DNA damage due to improved DNA repair mechanisms because of which less DNA is coming out of the nuclei. When the tail length is shortened after Yoga intervention, the % of DNA in the head was seen to increase. And vice versa, when the tail length increased after intervention, percent of DNA in the head was decreased.

Comet assay follows the principle of electrophoresis. Longer the tail lengths result from damaged DNA that has dropped out of the chromosome. So, in theory there can be 2 types of fragments resulting from damage dropping out, long and short. Long fragments are slow, moving while shorten ones migrate faster and further in the gel. So, when two2 samples (for pre and post) are compared where the tail length has shortened, it can be either due to lessened DNA damage in longer fragment or can also be due to lessened number of shorter fragment through repair joining together to form long fragments.

Earlier, comparative studies were conducted at SVYASA molecular biology lab on DNA damage among diabetics and healthy subjects had shown Results showed that DNA damage in diabetic people is significantly high than compared to healthy subjects (unpublished data). To make certain the tool we were using and also to validate the findings, the distribution of the current data was analyzed

The data showed a normal distribution indicating that there was no anomaly in the population studied or the assessment.

Different kinds of DNA repair mechanism exits like nucleotide excision, nich translation etc. Different mechanisms work on different kinds of damaged. Presence of shorted or longer fragments of damaged DNA needs to be understood further from the perspective of the kind of DNA repair that gets better with Yoga. Yoga is a known relieved of stress. Stress causes several physiological changes one of that is increase in oxidative molecules, the scope of this study does not let us point to the mechanism that is getting better but gives a convincing proof of betterment of DNA damage due to Yoga. There are no previous reports of previous study has seen the impact of a week of Yoga IAYT intervention on DNA damage in diabetes before. Ours is the first study to report this and we have shown highly significant impact of a holistic Yoga intervention on DNA damage in as lesser as a duration as 1one week of intervention., we found that DNA Also this is the first finding on the pervasiveness deep relaxation brought about by Yoga through parasympathetic dominance working at sub-cellular level.

Reference

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