crossing[14]. Secondly, the available maternal resources go waste due to premature fruit abortion. Thirdly, the Lorikeet, with its flower-pecking behaviour, removes numerous flowers daily. Fourthly, *Amegilla* and *Apis* bees collect large amounts of pollen, although these bees effect pollination. Lastly, availability of low maternal resources during dry period for the rapidly growing offspring; and leafless state during flowering and fruiting phase support this. Natural fruit set may be enhanced by managing nectar-feeding *Xylocopa* bees only, as other bees are involved in pollen collection which limits pollen availability for the prospective pollinators. Passerine birds can be managed only by taking measures for the conservation of habitats where *G. arborea* occurs.

The ripe fruits of *G. arborea* are fleshy, yellow and single-seeded. Monkeys collect fruits from the trees and forest floor and eagerly devour them whole or in part without separating the seed. The seeds come out unaffected through their excreta. The egg-shaped stony seed with pointed tip towards one end appears to be an adaptation to easily pass through the digestive system of monkeys and also to survive well during unfavourable conditions. While in the digestive system, the seeds may get coated with certain enzymes or substances and after passing through the digestive system; such substances may have a protective role for seeds against attacks by fungi on the forest floor and thus eliminate seed dormancy[15]. Monkeys cover a daily distance of up to 1 km in the forests responding to temporal and spatial dispersion of fruiting trees and search out individual ones which are consistently used year after year[15]. Therefore, the study suggests that monkeys use *G. arborea* fruits consistently and contribute to seed dispersal.

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Increased frequency of sister chromatid exchanges in patients with type II diabetes

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Sister chromatid exchange (SCE) is the exchange of homologous stretches of DNA sequence between two chromatids. Though SCE *per se* is not known to have any harmful medical effects, the higher frequency is associated with certain pathological conditions. Since patients with diabetes mellitus (DM) are at an increased risk for various vascular and other complications, it is likely that the extent of DNA damage is much higher in DM as compared to healthy controls. The present study is aimed to know the frequency of SCE in type II DM as compared to controls. A study of SCE, chromosomal aberrations and cell cycle proliferative index (CCPI) has been carried out in 35 individuals (20 type II DM patients and 15 normal healthy controls) from their peripheral blood cultures. Significant increase in SCE has been observed in DM patients as compared to the normal controls ($P < 0.0001$). No chromosomal aberrations or CCPI changes have been observed in any of the studied subjects. These results indicate that increased frequency of SCE in diabetic patients could be an early marker of DNA damage and a predisposing factor for some diabetes-related complications. There

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is a need to study other markers of DNA damage in DM, in larger sample size.

Keywords: Cell cycle proliferative index, chromosomal aberration, sister chromatid exchange, type II diabetes.

IT is evident that the generation of reactive oxygen species (oxidative stress) may play an important role in the etiology of some of the diabetic complications[1]. This hypothesis is supported by evidence that many biochemical pathways strictly associated with hyperglycaemia can increase the production of free radicals. They can interfere with several functions and mitosis of the cell. Free radical can cause oxidative damage to DNA[1]. SCE involves breakage of both DNA strands followed by an exchange of whole DNA duplexes. It occurs normally in cells during mitosis. Whenever genotoxic agents damage the cellular DNA, the rate of sister chromatid exchange (SCE) increases[2]. Many studies have shown that oxidative stress induced by hyperglycaemia possibly contributes to the pathogenesis of diabetes and its complications[3,4]. Lin *et al.*[5] have shown increased plasma level of malondialdehydrate (MLD) and total thiols in type II diabetic patients as compared to control subjects. Study has further shown that diabetic patients harbouring 16189 T → C variant of mtDNA impair the ability of a cell to respond properly to oxidative stress and oxidative damage. This suggests that diabetic complications may be related to oxidative stress-induced DNA damage. However our knowledge about SCEs in type II diabetes is limited due to single study carried out so far[6]. Hence the present study was undertaken to assess the frequency of SCEs, aberrations and CCPI in type II diabetes mellitus.

A randomized selection of 20 type II diabetic patients attending endocrine OPD at Sheth V.S. General Hospital, Ahmedabad was made, with written consent. Demographic data, detailed clinical history and peripheral blood were collected from each individual. They were investigated for fasting and postprandial blood sugar (FBS and PPBS), various lipid parameters, fasting insulin, C-peptide and HbA_{1C} to see the glycaemic control. Further investigations like fundus examination, estimation of serum thyroid stimulating hormone (TSH), C-reactive protein (CRP) and detection of urinary micro-albumin (UMA), were also carried out.

Fifteen healthy volunteers, non-obese, non-diabetic and matched with the diabetes mellitus (DM) patients for age and sex were selected as controls whose fasting and post-lunch sugars were normal with a normal lipid profile. None of the controls had any systemic change.

All the biochemical analyses for glucose, lipids, insulin, HbA1c, etc. were carried out using commercially available kits and quality control was assessed by using Bio-Rad controls for all parameters. Inter- and intra-assay verification were 8 and 5% respectively.

SCE studies were carried out in 20 type II diabetic patients and 15 healthy subjects. Blood samples were collected in aseptic condition in a sodium heparin vacutainer and metaphase preparation was carried out from phytohaemagglutinin (PHA)-stimulated culture using RPMI- 1640 media. A thymidine analog, 5-Bromodeoxyuridine (BrdU) was added at a final concentration of 10 µl/ml at 24 h, which will cause incorporation of BrdU in replicating DNA for three successive cell cycles, and subsequently subjected to photo-degradation. Slides from individuals were stained by solid Giemsa before photo degradation to examine chromosomal aberration. To visualize the resulting SCEs and cell cycle proliferative index (CCPI), differential Giemsa staining was carried out according to Perry and Wolff[7], with slight modifications. All the slides were coded and blind scoring was carried out under light micro-

Table 1. Clinical and biochemical profiles of the type II diabetic subjects

Clinical descriptors		*N*
Obesity (BMI > 26.5 kg/m^2)		13
Glycaemic control ($HBA_1c \leq 7$)		9
Dyslipidaemia		11
Hypertension		16
Coronary heart disease		6
Family history:	(i) Type II diabetes	10
	(ii) Cardiovascular disease	2
	(iii) Hypertension	9
Thyroid disorder		3
Tuberculosis		2
On foliate/B12 supplementation		3

Biochemical profiles of type II diabetic patients

FBS (mg/dl)	No. of subjects	PPBS (mg/dl)	No. of subjects
≤ 110	4 (20%)	< 140	2 (10%)
> 110	16 (80%)	≥ 140	18 (90%)
Fasting insulin (µIU/l)	**No. of Subjects**	**HbA_{1C} (%)**	**No. of subjects**
≤ 20	14 (70%)	–	–
20–40	5 (25%)	≤ 7	9 (45%)
≥ 40	1 (5%)	> 7	11 (55%)
Total cholesterol (mg/dl)	**No. of subjects**	**TG (mg/dl)**	**No. of subjects**
≤ 200	13 (65%)	≤ 170	13 (65%)
> 200	7 (35%)	> 170	7 (35%)
HDL (mg/dl)	**No. of subjects**	**LDL (mg/dl)**	**No. of subjects**
< 35	2 (10%)	≤ 130	15 (75%)
≥ 35	18 (90%)	> 130	5 (25%)
CRP (mg/l)	**No. of subjects**	**UMA (µg/min)**	**No. of subjects**
≤ 5	16 (80%)	≤ 15	15 (75%)
> 5	4 (20%)	> 15	5 (25%)

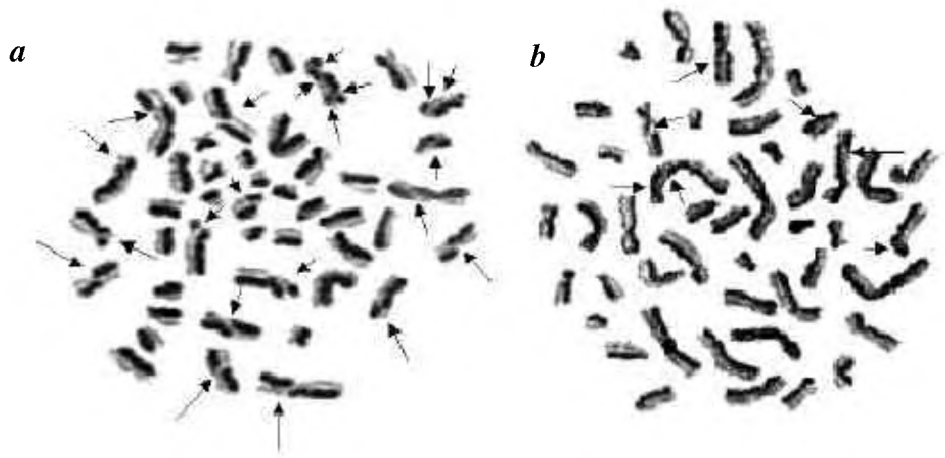

Figure 1. Metaphase in cell cycle II showing SCE frequencies in (*a*) type II diabetes case and (*b*) normal healthy control. Arrows indicate chromatid exchange.

Table 2. Frequencies of SCE in type II diabetes and normal healthy controls

	No. of subjects	No. of metaphases screened	No. of metaphases in M2 cell division	No. of metaphase mean ± SE
Type II diabetes cases	20	1693	752	7.81 ± 0.63*
Normal healthy controls	15	1507	596	6.18 ± 0.32*

*$P < 0.001$.

Table 3. Frequencies of SCE with age groups in diabetes cases and controls

Age group in years	SCE/metaphase in cases, $n = 20$ mean ± SE	SCE/metaphase in controls, $n = 15$ mean ± SE
< 40	7.44 ± 0.51 $N = 2$	5.84 $N = 1$
40–49	7.64 ± 0.67 $N = 7$	6.20 ± 0.28 $N = 6$
50–59	7.75 ± 0.64 $N = 5$	6.25 ± 0.18 $N = 4$
≥ 60	8.19 ± 0.57 $N = 6$	6.13 ± 0.29 $N = 4$

N, the number of subjects.

scope by a single individual. The influence on the cell cycle proliferative index was evaluated by recording the percentage of cells in the first (M1), second (M2), and third (M3) cell divisions by scoring at least 100 metaphases. A total of 25 cells in M2 phase with well-spread chromosomes from each culture were examined for SCE frequency. Statistical analysis of data was carried out using Student's *t* test.

The clinical and biochemical profiles of the diabetic patients are shown in Table 1. Hypertension and dyslipidaemia are found more common among diabetic subjects while none of the control subjects had either. In addition, 30% of the diabetic subjects had higher fasting insulin level. Mean age of the type II diabetic group was 52 ± 10.79 years and that of normal healthy control was 52 ± 10.25 years with mean BMI of 26.94 kg/m^2 and 26.49 kg/m^2 respectively. Duration of the disease varied from 6 months to 40 years.

Out of 20 diabetic patients, 9 (45%) have shown the glycaemic control, confirmed by results of PPBS and HbA1c. Eleven cases (55%) were found to have dyslipidaemia along with hypertension. In the diabetic group 10 (50%) patients have shown a family history of type II diabetes. Increased levels of serum, total cholesterol and triglycerides (TG) were observed in 7 (35%) patients whereas low level of HDL and high level of LDL were found in 2 (10%) and 7 (35%) patients respectively. In diabetic group, 4 (20%) patients have shown increased CRP and 5 (25%) have shown increased UMA values.

No chromosomal aberrations were observed in either of the study groups. The cultures from diabetic subjects and controls showed presence of cells in 1st, 2nd and 3rd cell divisions. The frequencies of sister chromatid exchanges in type II diabetic patients and normal controls are shown in Figure 1 *a*, *b* and Table 2. The mean value demonstrates the number of SCE observed in minimum 25 cell of second cell division (M2). There is a significant difference of SCE/metaphase between DM patients 7.81 ± 0.63 and controls 6.18 ± 0.32 ($P < 0.0001$). Table 3 shows the SCE frequencies with age groups in both cases and controls. No significant variation was observed in the CCPI of DM patients as compared to the controls. Table 4 demonstrates SCE frequencies in diabetic patients under various markers and risk factors of diabetes-related complications. There were no significant differences observed.

Table 4. Frequencies of SCE under markers and risk factors in the diabetic group

Glycaemic control	SCE frequencies	Obesity	SCE frequencies
Controlled	7.78 ± 0.74	Non obese	7.77 ± 0.70
Uncontrolled	7.84 ± 0.62	Obese	7.84 ± 0.56
Dyslipidaemia	SCE frequencies	Coronary heart disease	SCE frequencies
Absent	7.75 ± 0.70	Absent	7.90 ± 0.61
Present	7.86 ± 0.60	Present	7.60 ± 0.68
Serum CRP	SCE frequencies	UMA	SCE frequencies
Normal	7.79 ± 0.68	Normal	7.83 ± 0.62
Increased	7.89 ± 0.43	Increased	7.76 ± 0.74

Increased oxidative stress induced by hyperglycaemia plays a possible role in the pathogenesis of diabetic complications. Hence, many studies have been carried out to study an association of DNA damage with hyperglycaemia by means of mtDNA (mitochondrial DNA) mutations[8,9], increased urinary 8-hydroxy-2 -deoxyguanosine (8-OHdG)[10–13], leukocyte DNA damage measured as formamidopyrimidine DNA glycosylase (FPG)-sensitive sites[14]. The present study has shown increased SCE frequencies in type II diabetic patients as compared to the normal healthy control ($P < 0.001$).

In a cohort study of chromosomal aberrations and SCE frequencies in peripheral blood lymphocytes of a large population sample, Bender *et al.*[15] had observed a statistically higher mean frequency of SCE in females than in males with significant positive regression with age. Hedner *et al.*[16] studied SCE and chromosomal aberration in relation to age and sex and did not find correlation between the frequency of SCE and age, but older individuals had significantly more structural aberrations than younger. They also observed that the females had significantly more SCE as well as structural chromosome aberrations than males. In the present study no difference was found in SCE frequencies between sexes in any of the groups. However, an age-related increase in SCE frequency was observed in DM patients as compared to controls. Moreover, absence of chromosomal aberration in presence of increased SCE could be due to increased metabolic activation and oxidative stress induced by hyperglycaemia that may contribute to the pathogenesis of diabetic complications. It can be hypothesized that increased SCEs is an early reversible marker of diabetic complication that can be prevented by an early intervention of genoprotective drugs.

Moreover, somatic DNA damage has also been linked to various disease processes including coronary artery disease (CAD) particularly those who also had diabetes[17]. It was suggested that DNA damage representing part of the pathophysiology of the diabetic complication might also be a therapeutic target.

In addition to the above findings, the groups with glycaemic control and poor control as well as DM patients with dyslipidaemia and without dislipidaemia have also shown no significant difference in SCE frequencies between these groups. Consequently, similar SCE pattern has been observed in obese diabetic subjects as compared to non-obese diabetic subjects. Almost equal SCE frequencies were observed between patients with increased and normal values of CRP and UMA.

It is not desirable to over-interrupt DNA damage in diabetes as judged by SCEs, which may show reversible pattern by means of DNA repair[18]. But as evident from the study higher frequency of SCE in diabetic patients compared to controls would indicate a role of DNA damage in complications of diabetes, which needs to be studied in larger groups.

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Inundation characteristics and geomorphological impacts of December 2004 tsunami on Kerala coast

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Inundation characteristics and geomorphological changes associated with the December 2004 tsunami along the Kerala coast are presented here based on post-tsunami surveys, tide data and beach profile measurements. Maximum inundation and damage was caused in the zones adjoining Kayamkulam inlet in southern Kerala. Arrival of the tsunami coincided with the high tide in this sector, resulting in maximum devastation. Impact of the tsunami was not felt in the afternoon in northern Kerala due to the ebb or low tide and maximum inundation along that sector is reported during midnight, coinciding with the next high tide. Beach profile measurements confirm erosional tendency of the tsunami, which deposited huge quantities of black sand in the hinterland regions of the worst-affected Kayamkulam inlet area.

Keywords: Beach erosion, inundation, Kerala coast, run-up level, tsunami.

THE December 2004 Sumatra–Andaman earthquake generated a tsunami of unprecedented proportions, that devastated the shores of many Asian countries. The tsunami affected many parts of the Kerala coast[1], located in the shadow zone with respect to the direction of propagation of the tsunami, and in that sense its severity was rather unexpected. Nearly 200 people were killed and hundreds injured in addition to the loss of houses and properties worth several crores of rupees. Although there are reports of some previous tsunamis (1881, 1833, 1941, to mention a few), generated by earthquakes in the Andaman–Sumatra region, there is no documented evidence of any such events affecting the Kerala coast. A 1945 earthquake of M 8.0 in the Mekran coast is believed to have generated significant tsunami run-up in some parts of Gujarat[2,3], the only documented report of any tsunami affecting the west coast. To the best of our knowledge, the 2004 tsunami is the first of its kind to have affected the Kerala coast (Figure 1).

A significant observation associated with the 2004 tsunami effects along the Kerala coast is its localized amplification in some regions and totally subdued effect elsewhere. Understanding the spatial pattern of the tsunami and its effects on the coastal morphology has important implications for assessing future scenarios of inundation. Since most evidence left by tsunamis is perishable, it is important to carry out post-tsunami surveys to measure the run-up heights, inundation limits, arrival time of waves and assess the impact on the coastal life and property, flora and fauna, geomorphology, etc. Such information is important for future hazard assessment, and to develop inundation models. Tsunami-mitigation strategies have to be formulated based on such database[4]. Here, we report post-tsunami observations along the 560-km long stretch of Kerala coast as well as geomorphological changes in the shores of the Kayamkulam inlet region, where the effect of the tsunami was most severe. We use these observations to analyse the impact of the tsunami with respect to wave propagation and tidal characteristics of the region.

During the post-tsunami days, starting from 27 December 2004, field visits were conducted at different locations of the Kerala coast. The *Post-Tsunami Survey Field Guide* published in the website of International Tsunami Information Centre (ITIC)[5] was taken as a guide in the field trip. Sea level variations were studied from data available for Cochin and Neendakara (near Quilon). For Cochin, the tide gauge record available in the NIO website, which is based on data from the tide gauge at Cochin Port, was used. Sea-level data for Neendakara are observations by the Hydrographic Survey Wing of the Government of Kerala

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