SERUM VITAMIN E AND THE SICKLING STATUS
IN CHILDREN WITH SICKLE CELL ANAEMIA AS
SEEN AT KENYATTA NATIONAL HOSPITAL.

A dissertation submitted in part fulfilment
for the degree-of Master of Medicine (Paediatrics
and Child Health) Degree of the University of
Nairobi ~ .1985* "

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This dissertation is my original work and has not been presented for a degree at any other University.

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<table>
<thead>
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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>KNH</td>
<td>Henyatto National Hospital</td>
</tr>
<tr>
<td>PDU</td>
<td>Paediatric Observation Hard</td>
</tr>
<tr>
<td>SCO</td>
<td>Sickle Cell disease</td>
</tr>
<tr>
<td>SCA</td>
<td>Sickle cell anaemia</td>
</tr>
<tr>
<td>DNA</td>
<td>Deoxyribonucleic acid</td>
</tr>
<tr>
<td>HDL</td>
<td>High density lipoproteins</td>
</tr>
<tr>
<td>ISC's</td>
<td>Irreversibly 5'ickled cells</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td>A</td>
<td>Angstrom units</td>
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SUMMARY

In this study, serum vitamin E levels were determined among 62 children with sickle cell anaemia and 35 age-matched controls. Among the sicklers, the irreversibly sickled cell counts were done and correlated with the serum vitamin E levels.

There was a significant difference in serum vitamin E values ($P < 0.001$), the sickle cell anaemia patients showing tendency to deficiency with 27% of them deficient as against 20.5% of the controls.

It was found that the vitamin E deficient sicklers had a significantly higher irreversibly sickled cell counts ($P < 0.002$), indicating that vitamin E is an important inhibitor of the irreversibly sickled cell formation.
INTRODUCTION:

Sickle cell anaemia (SCA) is a genetic disorder caused by a point mutation in DNA (deoxyribonucleic acid) that codes for valine rather than glutamic acid, in the sixth position from the N-terminel, of the turn beta-globin chains of the haemoglobin tetramer. It occurs with high prevalsnc in tropical Africa and among emigrants from this region. The gene frequency on the continent ranges from and in Kenya from 0-25% among various tribes (1). It ha3 long bean known as a cause of morbidity and mortality in this region (2). Among patients attending the Kenyatta National Hospital (KNH) Paediatric Haeratology Clinic, subjects with sickle cell anaemia constitute two thirds of all cases (3).o

Since the first description of a case of sickle cell disease (SCD) by Herrick in 1910 (k) and the upsurge of research and literature, a lot has been uncovered as to the pathophysiology of the disease process in this condition. A large amount of work has gone into a-gt APKing to delineate the clinical features and pathophysiology in order to enable development of suitable and rational management of the condition.

The clinical manifestations of sickle cell anaemia are characterised by chronic haemolytic anaemia,
recurrent vasoocclusive painful attacks, frequent bacterial infections and, in some cases, eventual loss of organ function. However, a marked clinical diversity exists in this disease. For instance, some sickle cell anaemia patients have many vasoocclusive crises and require frequent blood transfusions and hospitalisations while others rarely have complications. Although there is no doubt that the molecular defect of sickle cell anaemia resides in the haemoglobin, additional secondary factors must be sought to explain these diverse clinical manifestations. One of these factors may relate to membrane peroxidative damage and its effects on the pathophysiology of sickle cell anaemia.

Tappel (5) suggested that peroxidative reactions contribute to the degenerative processes that eventually lead to cellular breakdown. Erythrocytes in circulation are especially prone to peroxidative damage because conditions that favour peroxidation are apparently optimal in these cells: possession of a membrane rich in polyunsaturated fatty acids, continued exposure to high oxygen tensions and acting as a vehicle for haemoglobin, one of the most potent catalysts for initiation of peroxidation (6). The fact that normal cells are protected from peroxidation in vivo is attributable to efficient antioxidant mechanisms:
partly a function of structural, integrity of each cell and partly reflective of the antioxidant systems within the cell. Included are superoxide dismutase, glutathione peroxidase, catalase and vitamin E. An impairment of any of these mechanisms may render the erythrocyte more susceptible to peroxidation eventually leading to its breakdown.

ChiUj Lubin and Shohet have demonstrated increased in vitro susceptibility of the red blood cells to peroxidation in sickle cell anaemia patients. This increased susceptibility is not entirely due to abnormal membrane lipid asymmetry. It has indeed been shown that even under oxygenated conditions in which most red blood cells containing sickle haemoglobin are biconcave discs, such erythrocytes are still more susceptible to lipid peroxidation than are normal erythrocytes. This suggests an abnormality in the antioxidant system in addition to that Induced by abnormal membrane lipid asymmetry. Superoxide dismutase, which provides protection for cytoplasmic components against damage by superoxide radicals, was reported by Nair, McCullough and Das (a) to be elevated in sickle cell erythrocytes. It has also been shown that erythrocyte glutathione peroxidase activity elevated in sickle cell anaemia patients and that there is a significant negative correlation between erythrocyte vitamin E and glutathione peroxidase activity in these patients.
The abnormally shaped erythrocytes present in sickle cell anemia patients regain their normal biconcave shape upon reoxygenation. However, a portion of the circulating red cell retain their sickle shape even when fully oxygenated; the irreversibly sickle cells (ISCs) are the number of irreversibly sickled cells are relatively constant in the same individual although they may vary from among patients (10, 11, 12). The haemolysis observed in sickle cell anemia subjects correlates well with the percentage of irreversibly sickled cells (11).

There is evidence that the formation of irreversibly sickled cells is related to changes in the membrane rather than alteration in the haemoglobin structure (13, 14, 15). During the sickling process, there is net loss of membranes as evidenced by membrane fragmentation demonstrable by cinematography (15), rearrangement of membrane lipids (7), changes in membrane-haemoglobin interactions (13) and increased potassium and water loss (16). It has been suggested that a defect in 'spectrin-actin lattice' may be the abnormality of the irreversibly sickled cell membrane accounting for the permanent bizarre shapes of these cells (10).

All the vitamin E in the erythrocytes is located in the membrane (17). In vitro studies have shown that
the red blood cells of persons deficient in vitamin E are more prone to oxidation (16, 19), more susceptible to peroxiriative haemolysis (19), more readily form Heinz bodies after exposure to hydrogen peroxide (20) and are more susceptible to potassium loss (21). Vitamin E apparently stabilises the erythrocyte membrane against oxidative stress probably functioning as a free radical scavenger (22) and also structurally stabilising the erythrocyte cell membrane (23).

It has been shown that vitamin E equilibrates rather rapidly with red blood cells (24), hence serum vitamin E levels are a good reflection of the red blood cell levels.

The foregoing stimulated the author to evaluate the serum vitamin E and the Sickling status among children with sickle cell anaemia in order to determine its contribution to the pathophysiology of this condition.
AIMS AND OBJECTIVES:

1. To determine and compare serum vitamin E levels in children with sickle cell anaemia (haemoglobin SS) and age-matched controls (haemoglobin AA).

2. To determine whether the serum level of vitamin E correlates with the sickling status as determined from the irreversibly sickled cell counts.
MATERIALS AND METHODS:

This study was conducted at the Paediatric Haematology clinic of the Henryatta National Hospital from October 1383 to January 1984. The clinic is held on Monday mornings and handles the bulk of children with haematological problems that reside in the city of Nairobi, but also handles haematological referrals countrywide. Host children confirmed by haemoglobin electrophoresis to have sickle cell anaemia that live in or around the City are followed up in this clinic. Most of these children are in steady state and are seen once every 2 to 2 months, the severe forms being seen more often.

On each visit, the parents are interviewed about the general health of the child at home since the previous visit and the child reappraised particular attention being paid to fever, jaundice, pallor of mucous membranes, or features of vasoocclusive painful attacks. Those with Harked pallor of mucous membranes and who on coultergram have a haemoglobin concentration less than 5g/dl, those with severe vasoocclusive painful attacks or with evidence of severe bacterial or other infection are admitted to the hospital's Paediatric Observation Ward (POD!) for appropriate care. The children in steady state are maintained on daily folate acid and proguanil usually obtaining enough stock to last them until the date of the next appointment.
DEFINITIONS:

The following definitions and criteria were used for purposes of this study:

1. Sickle cell anaemia (SCA) indicates homozygous inheritance of the sickle cell gene (haemoglobin SS). All red cells in these patients contain sickle haemoglobin.

2. Normal vitamin £ levels were taken as 5-20 micrograms per ml; levels below this range reflect deficiency (25).

3. The irreversibly sickled cell count is the percentage of sickled cells after equilibration of the blood with oxygen to full oxygenation.

Clinical Methods:

In the study period, 5 children on follow up for sickle cell anaemia who were the earliest to register at the clinic on each clinic day were entered into the study provided they satisfied the following:

1. Had no clinical evidence of crisis: hyper-haemolytic, vasoocclusive or severe infection,

2. Had not had a blood transfusion in the preceding 3 months.

3. Were not on medications known to affect liver function e.g. phenothiazines, barbiturates.

k. Were above the 80% line of the 50th centile of the Harvard standards for...
For controls, age-matched children attending the general Paediatric outpatient clinic for senior problems were used.* This is a busy clinic running every day from 8.00 am to 5.00 pm. The author attended the clinic on Wednesday mornings, whenever possible, to see children alongside the medical officers working in the unit. Every second child lined up for the author would be entered into the study provided they satisfied criteria 2, 3 and k as for sicklers, and had haemoglobin AA on haemoglobin electrophoresis.

For each of the children recruited into the study, the author took record of name, sex, a brief history including frequency of crises, hospitalisations and date of last blood transfusion. He then carried out a clinical examination and, took record of the nude body weight. After obtaining informed consent, 7.5 ml of blood was drawn by venepuncture under aseptic conditions and divided into 2 portions—

1. 2 ml was put into a sequestrene bottle which was gently mixed and labelled. This was destined for peripheral blood film for reticulocyte and white blood cell differential counts, irreversibly sickled cell counts, estimations of haemoglobins F and A, o
2. 5.5 ml was placed in a universal battle
covered with aluminium foil to prevent photo-
degradation. This was deep frozen at -20°C
until vitamin E assays were done.

Laboratory Methods:

The laboratory tests and essays were done by competent
laboratory technologists in haematology and chemical
pathology in the Nutrition laboratory of the Medical Research Centre, Kenya Medical Research Institute,
Nairobi.

1. Haemogram was done by use of Coulter counter
Model 'S'.

2. The haemoglobin was characterised by electro-
phoresis on cellulose acetate paper at pH 8.6.

3. Thin blood films were prepared and stained
with Jenner-Giemsa stain and reticulocyte
and white blood cell differential counts done
by light microscopy.

4. Haemoglobin F was measured by alkali
38°
denaturation method (26).•

5. Haemoglobin ft was measured by the
cellulose acetate elution technique.,

S« The irreversibly sickle cell count urns done
using a modification of the method of Gerties
find-Milner (27) • 0.5 ml of anticoagulated
blood UBS spun at 15DD rpm, cells and plasma were reconstituted to haematocrit of 25% and equilibrated with 95% oxygen and 5% carbon dioxide for 1D minutes. Very thin blood films isjere immediately prepared and stained by the Jenner-Giemsa stain and the irreversibly sickled cell count determined by counting 500 red blood cells by light microscopy.

Serum vitamin E uas determined using the Aminco-Bouian spectrophotofluorometer after the method of Hansen and Warwick (2B>«

Clinical and laboratory data were recorded on the patient’s data sheet appendix I.

Statistical Methods:

Student’s 't' test for paired comparison and the conditional test on means were used®
RESULTS: —>TYI PF NAIKOBI

A total of 62 sicklers and 35 controls were entered into the study. Among the sicklers the irreversibly sickled cell count was possible in h9. The complete data on various indices are presented in appendices IIA and XIB.

Figure 1 shows the scatter of the serum vitamin E levels among sicklers as compared to the controls. Also shown in the figure are the 95% confidence intervals for patients and controls. The bands are evidently distinct depicting in a picturesque manner the significant difference that exists in the 2 groups of children.

Among the sickle cell anaemia children 17 out of 62 (27.5%) had values of serum vitamin E in the deficiency zone whereas in the controls only 1 out of 35 (2.9%) was in the deficiency zone.
FIGURE: I p j ; A i r U M -1/cTAf, IN [el! LEUELY

T'ITICKLERS AND CONTROLS'; ' """ U M T r '':

; 5CA' children = Corit'faia

18

16

A#! I » I

9

ES v = 9" cQr, fidence interval for control::

' ": "i

'... I.confidence intf?rval for SC":.x:hil. «n

•vp: uc:' beloilli dotted line reflect deficiency.
Figure 2 shows the relationship between serum vitamin E and age in a scatter diagram. 95% confidence intervals are likewise indicated for sicklers and controls. There is no significant variation of serum vitamin E with age among both the sicklers and controls.
Figure 2 shows the relationship between serum vitamin E and age in a scatter diagram. 95% confidence intervals are likewise indicated for sicklers and controls*. There is no significant variation of serum vitamin E with age among both the sicklers and controls.
FIGURE 2: Serum Vitamin E Levels.

Legend:
- CONTROL
- JJA Patient

Confidence interval for controls
95% confidence interval for 5CA patients

Values below the dotted line reflect deficiency.
### Table I: Vitamin E Levels in the Sickle Cell Anaemia Patients and Controls

<table>
<thead>
<tr>
<th>SUBJECTS</th>
<th>Range</th>
<th>Mean ± ISO</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCA patients</td>
<td>0.4 - 12.5</td>
<td>6.06 ± 2.91</td>
</tr>
<tr>
<td>Normal controls</td>
<td>4.0 - 19.4</td>
<td>11 ± 4.14</td>
</tr>
</tbody>
</table>

In the population of 32 patients, ranging from age of 6 months to 12 years the serum vitamin E levels varied from 0.4 to 12.5 micrograms per ml with a mean of 6.06 ± 2.91. The range for the 35 age-matched controls was 4.0 - 19.4 micrograms per ml with mean of 11 ± 4.14. The difference in means is statistically significant (P<0.001).
Table 2: FREQUENCY DISTRIBUTION OF VITAMIN E LEVELS IN THE SICKLE CELL ANAEMIA PATIENTS AND CONTROLS

<table>
<thead>
<tr>
<th>VALUE</th>
<th>CLASS</th>
<th>PATIENTS</th>
<th>CONTROLS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mg/ml</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0-19</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2-3.9</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>4.9-5.9</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>6-7.9</td>
<td>6k</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>8-9.9</td>
<td>a</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>10-11.9</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>12-13.9</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>14-15.9</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>17</td>
<td>16-17.9</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>19</td>
<td>18.9-19.9</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>20-21.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>62</td>
<td>35</td>
<td></td>
</tr>
</tbody>
</table>

\[ X \bar{b} = 6.06 \]
\[ \text{S}X \]

Table 2 shows: the frequency distributions and the statistical analysis. P< 0.001 and the 99% confidence interval of difference 2.90 - 7.20
Table 3: THE IRREVERSIBLY SICKLED CELL COUNTS IN VITAMIN E DEFICIENT SICKLERS AS COMPARED TO SICKLERS WITH NORMAL LEVELS

<table>
<thead>
<tr>
<th>Vitamin E</th>
<th>&lt;5Mg/ml</th>
<th>≥5h g/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>17</td>
<td>32</td>
</tr>
<tr>
<td>Range ISC</td>
<td>2.2-28</td>
<td>0.2-19.2</td>
</tr>
<tr>
<td>Mean</td>
<td>9.37</td>
<td></td>
</tr>
<tr>
<td>Mean of transformed data</td>
<td>2.85</td>
<td>2.02</td>
</tr>
</tbody>
</table>

T value of difference 2<65

P <0.02

Table 3 shows a comparison of irreversibly sickled cell count among children with sickle cell anaemia who had normal values of serum vitamin E and those who showed deficiency of this vitamin. There is shown to be a statistically significant elevation of the irreversibly sickled cell count in vitamin E deficiency (P <0.02).
The whole blood haemoglobin concentration ranged from $5.1 \times 10^{-10}$ g/dl with a mean of 8.0 g/dl. There was a slight positive, but not statistically significant correlation ($r = 0.60$), between serum vitamin E and whole blood haemoglobin concentration. This means that the vitamin E deficient patients tended to be more anaemic. There also was a slight negative, but not statistically significant correlation ($r = -0.38$), between serum vitamin E and the reticulocyte count. This means that the vitamin E deficient children tended to have higher reticulocyte counts, an evidence of increased haemolysis in these children.

The range of sickle haemoglobin among 55 children for whom it was determined, was 77.2–96.2% with a mean of 81.7%. The range of haemoglobin F was $1.5\%$ with a mean of 7.0%. Haemoglobin $A^\text{s}$ ranged from 0.5–30% with a mean of 1.7%. 
DISCUSSION:

The results of this study show that children with sickle cell anaemia have significantly low serum vitamin E levels. The 62 children with sickle cell anaemia had serum vitamin E levels that ranged from 0.00 to 12.5 micrograms per ml, with a mean of 0.05 ± 2.9 (27%). Some of these children had levels of vitamin E that fell in the deficiency zone. The 35 age-matched controls, on the other hand, had serum levels of vitamin E that ranged from 0.00 to 19.5 micrograms per ml with a mean of 11.1 ± 1. The difference in the means is statistically significant (P<0.001). These results are in agreement with those of Chiu and Lubin; although these investigators had only 16 patients in their study and analysed erythrocyte levels of vitamin E rather than serum levels.

The aetiological factors leading to low serum vitamin E levels in patients with sickle cell anaemia are not clear. However a number of possibilities exist. They include reduced intake, poor absorption and increased utilisation.
Since controls in this study were selected from the same catchment area for Henyatta National Hospital, there is no reason to believe that the differences in vitamin E levels reflect differences in dietary intake as it is presumed that these groups are within the same social class and their eating habits are similar.

Rosenblate (29), Bogoch (30) and Song (31) in their various papers showed that subjects with sickle cell anaemia suffer marked hepatic dysfunction as shown by altered liver function tests pointing to parenchymal damage, biliary and canalicular stasis and impaired bile salt excretion. In the absence of bile salts, the absorption of vitamin E is markedly impaired (32).

In a recent study, Hinoti, Ndombi and Kyobe (33) have observed decreased serum levels of high density lipoproteins (HDL) in the serum of children with sickle cell anaemia as compared to normal controls. This observation is similar to observations made for cystic fibrosis patients (34). Since HDL is known to be important in vitamin E absorption (35), and it has been suggested that HDL-vitamin E is probably the
metabolically important form of the vitamin^ with HDL being able to transfer and accept vitamin E from red blood cells to a greater extant and at a faster rate than other lipo-proteins (35) low vitamin E levels in serum of sickle cell anaemia patients may be secondary to HDL deficiency in these subjects. HDL deficiency may result from liver dysfunction.

Another contributing factor to the low vitamin E levels may be increased utilisation in serum. Haemoglobin, as found in plasma in sickle cell anaemia patients as a result of intravascular haemolysis, Is known to be a powerful catalyst of lipid peroxidation (6). The plasma haemoglobin might therefore catalyse the oxidative destruction of vitamin E and account for lower serum vitamin E levels in sickle patients.

The results of this study also showed that vitamin E deficient sicklers had higher irreversibly sickled cell counts than the sicklers with normal levels. Among the 49 patients in whom the irreversibly sickled cell count was done, the 17 (34%) who had deficiency of vitamin E had irreversibly sickled cell count ranging from 2 to 28% with a mean of 31%. The remaining 32, whose values for vitamin E
fell In the normal range had irreversibly sickled cell count ranging from 0.2-19.2% with mean of The difference in means of these -2 groups of aicklers was statistically 'significant CP < 0.02).

Structural abnormalities such as increased numbers of distorted and contracted cells have been found in vitamin E deficient premature infants (36). Shortened red cell survival has been reported in vitamin E deficient udults (37), vitamin E deficient premature infants (35) and cystic fibrosis patients with low blood vitamin E levels (37). The haematological abnormalities observed in these vitamin E deficient states can be corrected by vitamin E supplementations (36, *37, 38). Taken together, these findings suggest that enhanced susceptibility of sickle cell erythrocytes to peroxidation may be a factor that contributes to shortened lifespan of the red cell characteristic of sickle cell anaemia.

In addition to accelerated red cell destruction, peroxidative damage may play a significant role in the pathogenesis of irreversibly sickled cells. As earlier stated, the irreversibly sickled cell in morphologically identified by its failure to regain its biconcave disc shape when fully
oxygenated end is mechanically identified by its entrance fragility and rigidity.
Although there is no correlation between percent irreversibly sickled cells in peri-
pharal blood and vasoocclusive episodes Serjeent cr, ai (11) shewed direct relationship between percentage of circulating irreversibly sickled cells and extent of haemolysis.

The mechanism of irreversibly sickled cell formation is presently unknown. Current concepts of irreversibly sickled cell formation focus on irreversibly deformation of spectrin-actin lattice—the membrane skeletal proteins of the red cell—and on cellular dehydration secondary to abnormal membrane permeability. Studies by Lux, John and Harnorsky (1*0 indicate that this deformation is not dependent upon the persistant interaction between sickle haemoglobin and the spectrin-actin lattice. These investigators suggest that the irreversibly sickled cell formation is the result of perranent alteration in the spectrin-actin lattice. On the other hanri, studied by Clsrk, Mohandas and Shohet C^G) indicate that reduced defamiability of the irreversibly sickler cell is mainly due to dehydration of these cells and that the abnormal deformability can he returned
to normal following cellular rehydration,

The findings of these investigators imply that the inability of the irreversibly sickled cells to return to their original biconcave shape is due to a high internal viscosity which is resultant from dehydration caused by abnormal membrane permeability.

These two theories do not have to be mutually exclusive. It is possible that peroxidation damage can lead to permanent alteration of the spectrin-actin lattice as well as in abnormal membrane permeability. Possibly, the structurally normal spectrin-actin lattice network is passively deformed by oriented haemoglobin S microfilaments later becoming fixedly deformed. In the case of abnormal membrane permeability, it is possible that peroxidative damage effects membrane components required to maintain normal permeability. Such damage could alter cation transport, create 7QA holes in the membranes as suggested by Jacob and Lux (E+I) and lead to abnormal passive ion transport. A comparison of several properties of vitamin E deficient red cells following peroxidant injury with those of irreversibly sickled cells may provide some support into this view.
If vitamin E plays a role in inhibition of formation of the irreversibly sickled cells, then vitamin E supplemental ion to sickle cell anaemia patients should reduce the irreversibly sickled call counts in the peripheral blood. This is exactly what Natte, Mechlin and Brin (2,3) and Kinoti, Heme, Buibc and Daws (42) found in their etodies* A double blind crossover study is currently under way at the Paedistric Heematology clinic to evaluate the role of vitamin E supplements on the irreversibly sickled call count in sickle cell anaemia patients.

The results of this study confirm that peroxidsnt damage secondary on vitamin E deficiency, is a significant factor in the pathogenesis of the irreversibly sickled cell formation and hence the pathophysiology of sickle cell anaemia.
CONCLUSIONS:

From the results of this study the following inferences are noted:

1. It was established that there was very significantly low levels of vitamin E in children with sickle cell anaemia as compared with normal age-matched controls. Possibilities of this increased deficiency point to malabsorption and overutilisation.

2. Vitamin E deficiency was correlated with significant elevation of the irreversibly sickled cells. It can be inferred that vitamin E is an important inhibitor of irreversibly sickled cell formation.
RECOMMENDATIONS:

1. Sickle cell anaemia patients and their parents should be given instructive Nutritional Education to improve vitamin E intake and possibly be supplemented to provide minimal daily requirements.

2. A study to evaluate the interaction of vitamin E and other factors known to affect the pathophysiology of sickle cell anaemia, such as zinc and fetal haemoglobin levels.
ACKNOWLEDGEMENTS:

1. To my supervisor Dr. S.N. Hinoti whose advice was always invaluable through the whole study's much thanks.

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The Kenya medical Research Institute (K.E.M.R.I.) for availing their laboratory facility to me for the study, thanks.

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6. To Mr. Gemert, statistician at the Medical
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7. To Mrs. J. Thairu who patiently did the typing of this work, sincere gratitude.
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APPENDIX I - PATIENT'S DATA SHEET:

DATE: Create date

NAME: Carnella Comerio

SEX: Female

DATE OF LAST SEVERE ORIS: November 10, 2000

DATE OF LAST HOSPITALISATION:

DATE OF LAST BLOOD TRANSFUSION:

PRESENT MEDICATIONS UPTO 2 WEEKS PREVIOUSLY:

1. Xeloda 500 mg PO BID
2. Tamoxifen 20 mg PO QD
3. Bicalutamide 150 mg PO QD

HEMOGLOBIN CONNL: 12.5 g/dL
RED BLOOD CELL COUNT:
RED BLOOD CRIT:
LDH COUNT:

HAEMOGLOBIN F

HAEMOGLOBIN S

VITAMIN E
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