Report and Opinion

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(4) Introduction.
(5) Materials and Methods.
(6) Results.
(7) Discussions.
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Report and Opinion

Cover Page, Introduction, Contents, Call for Papers, All papers in one file

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The Reason Why Planets And Moons Move In The Same Direction

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Abstract：The main features of our solar system are that all the planets revolve around the sun in the same direction, as do most of their moons, and all the planets lie more or less in the same plane of the sun's own rotation. For centuries scientists have tried to find a theory for the solar system origin that can explain these features. Any theory that could not explain these features was usually rejected. But through mechanical analysis, the author of this article found that these main features are not the result of our solar system's origin as most theories hypothesized but that of mechanics of their movement. The author found that if a planet does not revolve in the plane of the sun's own rotation and circle the sun counterclockwise, it will not be stable and its course will be deflected. The moving pattern of our solar system determines these main features. Only in this moving pattern can the solar system survive. [Report and Opinion. 2009;1(1):1-5]. (ISSN: 1553-9873).

Key Words: the Sun, the Origin of the Solar System, the Coriolis Force

Up to now, theories for the origin of our solar system have tried to explain its main features that all the planets revolve around the sun in the same direction, as do most of their moons, and all the planets lie more or less in the same plane of the sun's equator. Most theories can explain these features well but can't adequately explain some other chemical or physical phenomena. In this article I will prove that these features are not the result of our solar system's origin but that of mechanics of their movement .

Through mechanical analysis, we will find that from whatever direction an alien celestial body intrudes upon our solar system, if it is captured by the sun or by a planet it will keep changing its orbit till it circles in the same direction and in the same plane as the already existing planets and moons do.

To simplify the discussion, let's take a look what will happen if the moon is not circling around the earth the   way it does now. (Let's call the direction in which all the planets circle around the sun counterclockwise, with the opposite moving direction clockwise.)

Suppose the moon is orbiting the earth clockwise to start with, but because the earth/moon system is moving around the sun counterclockwise, the vertically moving moon will be deflected by the Coriolis force([1]) and its orbit around the earth will tilt rightward, eventually becoming a counterclockwise revolution. The Coriolis force will continue deflecting the moon until its orbit overlaps the plane of the sun's equator. Ultimately the moon will circle the earth counterclockwise in the plane of the earth's orbit as it almost does now.
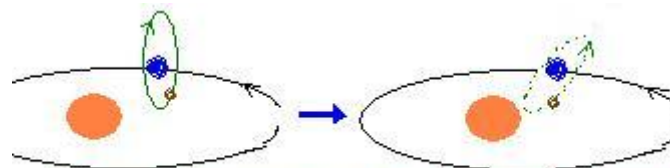
clockwise vertical orbit will tilt rightward

Now, let's suppose the moon's orbit around the earth is vertical to the plane of the earth's orbit and the direction is counterclockwise.

counterclockwise vertical orbit will tilt leftward
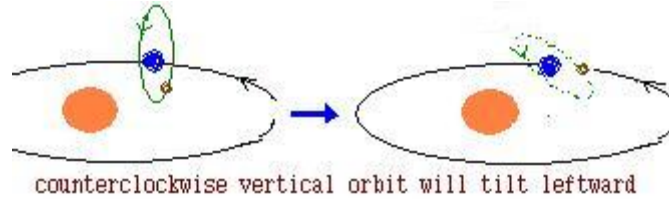
As illustrated above, the moon will be deflected by the Coriolis force and its orbit around the earth will tilt leftward to become counterclockwise around the earth eventually, too. The Coriolis force will continue deflecting the moon until its orbit overlaps the plane of the sun's equator completely. Ultimately the moon will also circle the earth counterclockwise in the plane of the earth's orbit.

Then, what if the moon was captured but clockwise by the earth right in the plane of the sun's equator to start with? By mechanical analysis, we can see that in this situation either the moon will collide with the earth or its orbit be distorted to become counterclockwise.

We know that when the moon moves around the earth, it is also moving around the sun. Between the moon and the earth, their masses, gravitational force, distance and the moon's relative orbital velocity to the earth must meet the Newton's law of universal gravitation and the circular motion principles. In addition, between the moon and the sun, their masses and motion parameters must also meet the Newton's law of universal gravitation and the circular motion principles. The relative orbit velocity of the moon can be calculated with the following formula.

$$ V = \sqrt{\frac{F \cdot R}{m}} $$

Where:
V -the relative orbital velocity of the moon
F -the resultant force that dominates the moon's motion
R -the distance between the moon and the sun or the earth
m -the mass of the moon.
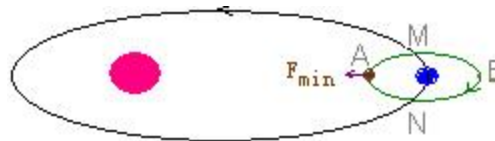
To simplify the problem, let us take four representative points A,B,M and N in the moon's orbit for analysis. When the moon is at point M or N, its relative orbit velocity to the sun is the same as that of the earth. As the moon moves clockwise further to point A, its relative orbit tangential velocity to the sun increases to the highest, but the forces from the sun and the earth are in opposite directions so that the resultant force (F) on the moon is at a minimum. Now the moon is at   a minimum distance to the sun (R), too. According the equation above, the moon should have a minimum relative orbital velocity to the sun (V). But the actual value is at maximum.

When we take point B for analysis, we will also find the inconsistence where the moon should have a highest relative velocity to the sun but the actual value is the lowest.
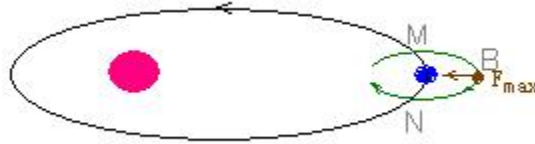
From the above analysis, we can see if a moon runs but clockwise around a planet, its orbit would not be stable. By further analysis we can see its orbit will be elongated to a narrow strip by and by and the collision possibility between the moon and its planet is very high or is inevitable.

However a moon comes to be captured by a planet, the moon can only move stably around the planet counterclockwise, the same as the way our moon is moving around the earth.

The above analysis accounts not only for the orbits of the moons around their planets but also accounts for the planets' orbit and the periodic comets around their suns. The principle is the same. The sun is moving around the Milky Way Galaxy center at a very high relative velocity. Any captured planet or periodic comet that doesn't move within the plane of the sun's equator will be deflected by the Coriolis force and be made to run counterclockwise around the sun within its equator plane. So the main features that the planets all revolve around the sun in the same direction, as most of their moons do, and the planets all lie more or less in the same plane of the sun's own rotation are not the result of the formation of the solar system, but is the result of the dynamics of our solar system.

Date: 2005-7-18

Notes:
(1).Coriolis force is a sidewise force exerted on a body when it moves in a rotating reference frame. It is a fictitious force because it is a by-product of measuring coordinates with respect to a rotating coordinate system as opposed to an actual push or pull.

PS
1. Some are suspicious of the Coriolis Force effect, and argued with me on Coriolis Force fiercely. So I reexplain my point in another way. Take the second illustration for example.

counterclockwise vertical orbit will tilt leftward
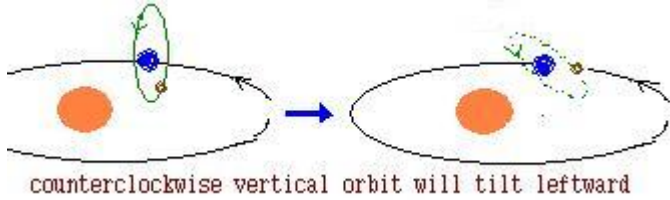
Let's see what will happen if the moon circles the earth freely in a counterclockwise polar orbit.

When the moon is right over the earth's north pole, its velocity (V) relative to the sun is the same as that of the earth because they are at the same orbital distance to the sun. Now the moon circles in, its centripetal force (F) to the sun is reduced. Why, because part of it is balanced off by the earth on the opposite side. On the other hand, its distance to the sun (R) is shortened now. Now let's use the formula

$$V = \sqrt{\frac{F \cdot R}{m}}$$

The F and the R are both reduced, but its velocity is the same as the earth's. So once the moon moves in between the Sun and the Earth, the moon will have redundant velocity and will go faster than required by the F and R.

A track and field athlete knows this principle well. The lead runner always occupies the innermost tack while turning so he can keep himself ahead of the others.

Now the moon is in the inner track and with the same relative velocity to the sun, it will surpass the earth. Thus moon's orbit changes.

When the moon is ahead of the earth, universal gravitation force between the moon and the earth, and that of between the moon and the sun   will apply a resultant force that will slow the moon down; Only when the speed of the moon is reduced by this among can the V, F and R in the above formula be satisfied.

2.   I make a summary of what my article already provided,   what my article is expected to provide, and what I can further provide. I just don't want to disappoint all the visitors here.

-It provides a new idea(the Reason Why planets and Moons Move in the Same Direction) on an old topic.

-It provides a laconic deduction.

-It provides a qualitative analysis.

-It is expected to provide detailed deduction on the origin of the solar system, including how   the sun, the planets and their moons formed, what were they before they became spheres...

-It is expected to present the reasoning mathematically.

quantitative analysis is expected.

-My specialty is not math, physics, or cosmology. And I am not in research. So it's not easy for me to present quantitative analysis if I get no help from competent scholars. Aware of my weakness, I deliberately chose this title for my article. I think my article can be considered as a complete thesis for the title I use(the Reason Why planets and Moons Move in the Same Direction) .

-If you ask me how the birth of suns, their planets and moons come about, it's out of the topic that I want to discuss. As Darwin told us the reason why polar bears are white, but should you censure him for not answering the question why there are white polar bears on earth? Those are two different topics.

-In my reasoning, I must simplify all the complicated systems so that I can reason it with my brain, or I will have to recourse to the advanced theories or complicated formulas which are beyond my competence.

-So, what I have provided and that I would provide are all basically qualitative analysis, just as Copernicus did when he discovered our solar system is solar-centric, but gave no equation to show how that is achieved.

I do hope my work can be further carried on by competent scholars in quantitative ways.

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Occurrence, Distribution and Sources of Organochlorine Pesticides (OCPs) in Karst Cave

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Abstract: Despite the numerous researches on Organochlorine pesticides (OCPs) in China and in the world, information regarding the distribution of OCPs in Karst caves is extremely limited. Karst area has much ecological vulnerability and it is so easy to be contaminated. This paper presents results of a monitoring program conducted in Dayan cave, Guilin, China that was designed to characterize levels, trends and sources of pesticides in soil samples. Thirteen soil samples were collected and OCPs were analysed. Inside the cave a total concentration of OCPs (∑OCPs) detected was 29.659 ng/g with a mean value of 3.295 ng/g and ∑OCPs detected outside the cave was 74.108 ng /g with a mean value of 18.527 ng/g. ∑OCPs outside the cave was higher than ∑OCPs outside the cave. The concentration of Chlordane in OCPs was highest among all the OCPs with range of 0.12─13.253 ng/g and mean value of 3.93 ng/g. The next compound with high level was Heptachlor wich ranged from Non-detected (ND) to 2.465 ng/g with a mean value of 1.4 ng/g. The pollution of OCPs in soil comparing with other countries and other areas in China was light. The analysis of Dichlorodiphenyltrichloroethane (DDT) and Hexachlorocyclohexane (HCH) isomers showed that there is a fresh input of Dicofol and Lindane in the area. By calculating the ratios of Dichlorodiphenyldichloroethane (DDD) to Dichlorodiphenyldichloroethylene (DDE) we found that the degradation of DDT outside the cave was aerobic and the degradation of DDT inside the cave was anaerobic. [Report and Opinion. 2009;1(1):6-16]. (ISSN: 1553-9873).

Key words: Organochlorine pesticides, Karst cave, Guilin, China

1.Introduction

Organochlorine pesticides are a group of persistent organic pollutants (POPs) which are to be eliminated or reduced on their release into the environment in many countries. Because of their persistence in the environment, and biological accumulation through the food web, OCPs can cause environmental damage, and affect human health (Colborn et al, 1996) .Due to their volatility and persistence in the air; OCPs are subjected to long-range atmospheric transport (LRAT). Therefore, OCPs released in the tropical and subtropical environments could be dispersed rapidly through air and water, and tend to be redistributed on a global scale (Tanabe, 1991) .The origin and fate of OCPs in soils with different land use have been extensively studied in many countries. Although the usage of OCPs was phased out for decades, the elevated concentrations were still observed in many agricultural soils (Harris et al., 2000) and the relationship between sites of greatest application and current residue levels was found strong (Shivaramaiah et al., 2002). The release of OCPs from soils continues to be a source to the environment (Meijer et al., 2001).

China is a large producer and consumer of Pesticides in the world (Rongbing et al., 2006). Large amount of OCPs were used in past decades to sustain over population in China. HCH and DDT were widely used in China from 1952-1983. Although their use had been discontinued in China since 1983, their persistence has left residual amounts in the soil in many areas (Zhao Ling

and Ma Yongjun, 2001). At present the use of DDT is still allowed to control mosquitoes, particularly in the malarial transmission zones in China (Zhang et al., 2005). Accordingly, China still produces a small amount of DDT and China is also allowed to export DDT to other countries for the same purpose. This paper presents the current status of OCPs residues in Dayan cave. The dataset generated will serve as a baseline for further studies.

2.Materials and Methods
2.1 Study Area

Region of research was in Guilin located in Guangxi Zhuang Autonomous Region in southeast China. Guangxi province (Southeast of China). The Geographical coordinates are 25° 40' 25" North, 108° 44' 0" East and has an altitude of 150m. It is bounded to the north-east by Hunan province, to the south-east by Hezhou town and it is next to Guangdong province. It has a surface area of 27, 800 square kilometers and a population of 4.76 million.

Dayan is an intermediate upper layer cave of Guilin Maomoatou cave system, located in the middle part of Guangming Mountain at right side of Taohua River in the north-west of Guilin. Guangming Mountain is a large peak cluster in Fenglin Plain, with an area of 0.92km^2, the highest peak altitude of 404.4m and the plain altitude of 151m.The outcrop is a thick limestone layer of the Devonian system with a high intensity of Karst process. Dayan is a noncommercial cave located northeast to Ludiyan cave and southeast to Guangming Mountain. The map of Guangxi showing Guilin and plane diagram of Dayan cave are shown in Fig 1 and Fig 2 respectively.
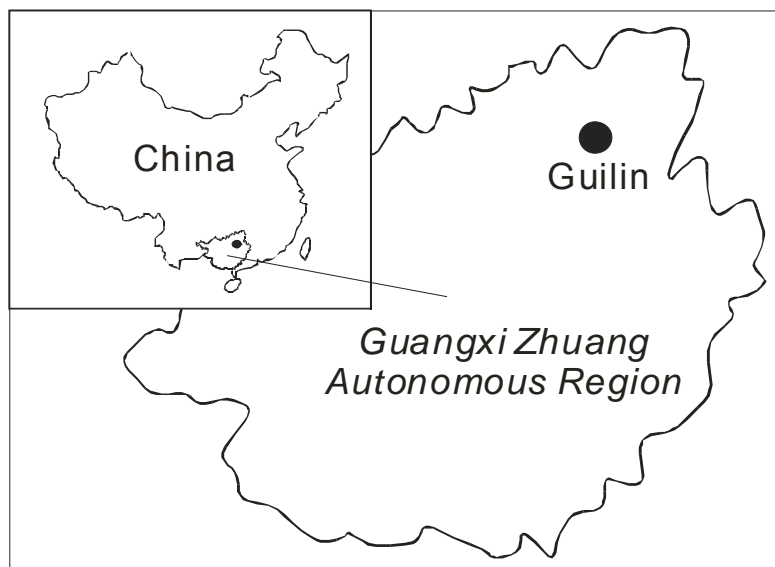
Fig 1: Map of Guangxi province showing Guilin
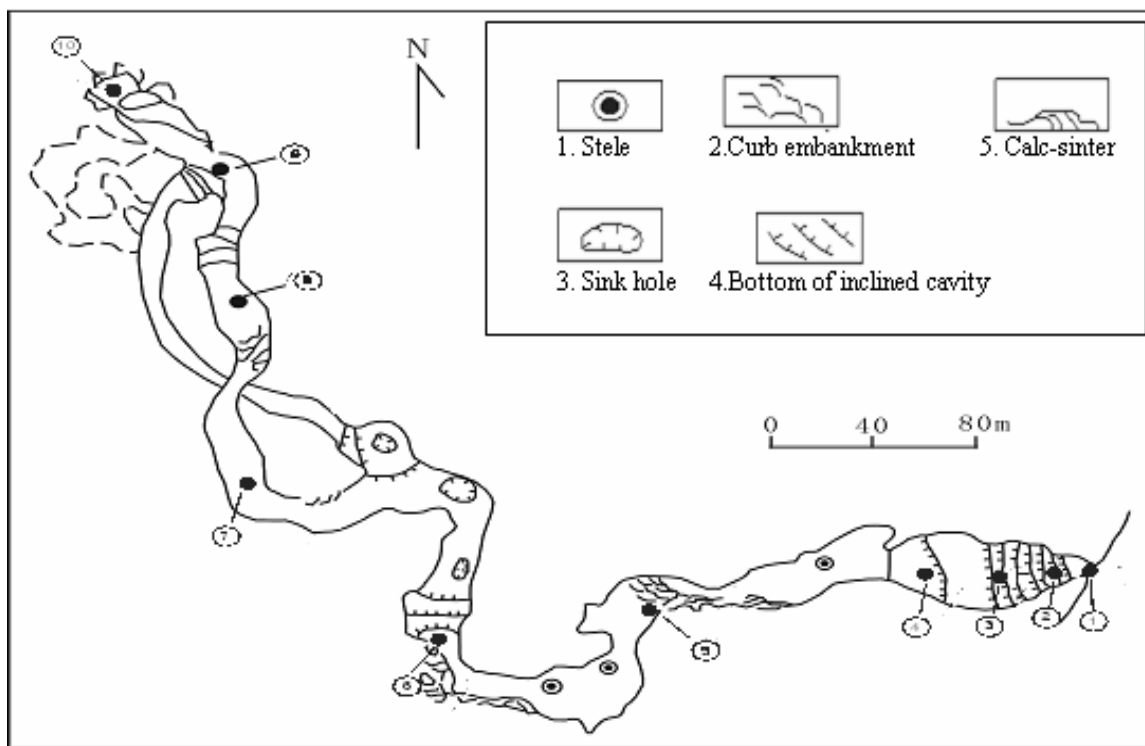
Fig 2: Map of Dayan Cave showing sampling locations (1─10) are sampling locations

2.2 Soil sampling

The collection of soil samples was done in September 2006 in the Dayan cave in Guilin. We placed 10 sampling locations inside the cave that followed the horizontal section from the east gate. (Sample number 1 was collected at the east gate) and the serial number was from 1-10 as shown in fig 2. Three samples (1 ', 2', 3') were also taken outside the east gate (downstream of the cave). Sampling was done with the use of a shovel and the samples were taken in a clean bag, and were well sealed and labeled. The weight of each sample was 500g. After the collection of samples, the samples were kept frozen in a refrigerator below -20°C prior to the commencement of the laboratory analysis.

2.3 Analysis
2.3.1 Experimental procedures

Before we began analyising the samples (before experiment) all glass wares were acid washed and cleansed with distilled water before they were dried in the oven at 200°C for about four hours. Reagents used for the experiment included: dichloromethane (DCM), hexane, acetone, sodium sulfate, alumina gel (100-200 mesh), silica gel (100-200 mesh), mesh hydrochloric acid and vitriol. Alumina and Silica gel were Soxhlet extracted for 24 hours in DCM solvent and then baked in the oven at 240°C and 180°C respectively for 12h. After cooling down, distilled water (3% of the reagent weight) was added to reduce the activity. Filter paper, aluminium foil, absorbent cotton and active copper were also used as materials.

A mixed standard sample of OCPs [2,4,5,6-tetrachloro-m-xylene (TCMX) and decachlorobiphenyl (PCB 209)] were purchased as surrogate standards and were added to all the samples before the extraction. The whole process of pretreatment was based on US EPA SW-8080A method as reference. 20 g of the sample were weighed with electronic balance and injected with the surrogate (using a syringe) before the sample was Soxhlet-extracted for 48 hours with redistilled Dichloromethane (DCM). Active copper slices were added to the conical flask containing DCM to eliminate the influence of sulphur contained in the sample. After 48 hrs in the soxhlet extractor, the extracted samples were added with Sodium sulphate ($NaSO_4$) to remove unwanted water. After that, the solvents were concentrated to about 5 ml and then passed though a mixture of silica gel and alumina gel (10/3, V/V) for purification and it was rinsed by a mixture of DCM and hexane (2/3,V/V). The solvent was then condensed with high purity Nitrogen. 4 ml of the hexamethyl-benzene and PCNB (5ppb) were added as internal standards to help in quantifying the amount of OCPs present in the samples. Finally samples were stored and kept in the refrigerator until next analysis (Analysis by HP 6890 GC).

2.3.2 Analysis by HP 6890 GC

HP 6890 GC (Gas Chromatography) was equipped with a ^{63}Ni electron capture detector and a 30 m x 0.32 mm i.d (0.25 *lm* film thickness) DB-5 fused silica capillary column. Nitrogen was added as a carrier gas at 1.2ml/min. the oven temperature was kept at $40^{o}C$ for 5 minutes and increased to $290^{o}C$ at a rate of $4^{o}C$/min. Injector and detector temperatures were maintained at 250 and $300^{o}C$ respectively. 2 Microliters (μl) of each sample was injected for analysis.

2.3.3 Quality control and Quality assurance (QC/QA)

Quality control and Quality assurance was made by the use of the USEPA method in the process of the experiment. The recovery rates and standard deviation of OCPs during separation and testing are within the limiting value of the US EPA 610 method. Recovery rates of TCMX and PCB209 are 69±6% and 76±7% respectively.

3.Results and Discussions
3.1 Concentration and distribution of OCPs

A summary of concentrations of OCPs detected in soil samples of Dayan cave is shown in Table 1.

Inside the cave ∑OCPs detected was 29.659 ng /g with a mean value of 3.295 ng /g and ∑OCPs detected outside the cave was 74.108 ng /g with a mean value of 18.527. ∑OCPs outside the cave is higher than the total concentration outside the cave (Fig 3). The levels of OCPs outside the cave compared to the levels inside indicate that despite the relatively closed environmental system of the cave and less human interference inside the cave, it still had OCPs contamination due to air transfer, rain water filtration and other processes, but the degree of contamination is not high.

Table1: Levels of OCPs in soil samples of Dayan Cave

	OCPs overall level range Min─Max(mean value）	OCPs level range inside the cave Min─Max(mean value) （2─10 samples）	OCPs level range outside the cave Min─Max(mean value) （1，1′，2′，3′samples）
α-HCH	0.014─0.170 (0.087)	0.014─0.126(0.043)	0.095─0.170(0.130)
β-HCH	0.026─0.219 (0.102)	0.026─0.219(0.087)	0.100─0.138(0.117)
γ-HCH	0.015─0.285 (0.092)	0.015-0.180(0.044)	0.075─0.285(0.140)
δ-HCH	0.009─0.072 (0.034)	0.009─0.045(0.024)	0.020─0.072(0.044)
TC	0.021─6.119 (1.841)	0.021─1.674(0.279)	3.226─6.119(3.403)
CC	0.085─7.134 (2.221)	0.101─3.111(0.849)	3.899─7.134(4.198)
Hep	ND─2.465 (1.399)	ND─1.087(0.139)	1.871─2.465(1.632)
Hep-Epo	ND─1.908 (0.911)	ND─1.022(0.379)	1.000─1.908(1.185)
EndoI	ND─0.230 (0.067)	ND─0.040(0.021)	0.103─0.230(0.122)
EndoII	ND─0.161 (0.046)	ND─0.057(0.021)	0.026─0.161(0.080)
Endosulfate	0.030─0.500 (0.175)	0.030─0.180(0.086)	0.200─0.500(0.294)
p,p'-DDE	0.011─0.342 (0.108)	0.011─0.109(0.041)	0.115─0.342(0.174)
p,p'-DDD	ND─0.121 (0.079)	ND─0.077(0.038)	0.011─0.121(0.059)
o,p'-DDT	0.049─0.467 (0.212)	0.049─0.226(0.113)	0.302─0.467(0.310)
p,p'-DDT	ND─0.090 (0.031)	ND─0.039(0.011)	0.046─0.090(0.050)
ΣDDTs[b]	0.094─0.875 (0.371)	0.094─0.384(0.162)	0.532─0.875(0.434)
ΣHCHs[a]	0.100─0.665(0.269)	0.100─0.453(0.197)	0.313─0.665 (0.430)
ΣOCPs[c]	1.159─23.625(10.911)	1.159─11.180(3.295)	13.250─23.625(18.527)

ND=Non- detected
ΣHCHs[a]= α-HCH + β-HCH + δ-HCH + γ-HCH.
ΣDDTs[b]= p, p'-DDE + p, p'-DDD + o, p'-DDT + p, p'-DDT.
ΣOCPs[c] =ΣHCHs+ΣDDsT+Σother OCPs.
Σother OCPs = Heptachlor (Hep) + Heptachlor epoxide (Hep-Epo) + TC (Trans-Chlordane) + CC (Cis-Chlordane) +EndoI (α-Endosulfan) +EndoII (β-Endosulfan) +Endosulfate.

   The amount of Chlordane (TC+CC) in OCPs was highest among all the OCPs detected inside and outside the cave with a total concentration of 39.689ng/g and mean value of 9.92 ng /g inside the cave and a total concentration of 4.52 ng/g outside the cave with a mean value of 1.13 ng /g.
   This is because South china have been using Chlordane to kill termites, so the high concentrations of Chlordane observed may be predominantly due to the use of technical Chlordane as a termiticide in this area in previous years . In China, technical chlordane is still being extensively used against termites in buildings, with an estimated amount of over 200 tons year^{-1} in recent years (Xu et al., 2004).
   The next compounds with highest levels were Heptachlor (Hep) and Heptachlor epoxide (Hep-Epo.) Heptachlor (Hep) was also used and produced in large quantity in China. From 1967 to 1969 the amount of Heptachlor produced was 17 tons, to kill the termites and other insects in the soil. It is shown in Fig 4 that the majors parts of OCPs (HCHs and DDTs) at the cave's innermost sampling points 9 and 10 did not show the lowest values, but rather slightly greater than the values of sampling points 7 and 8 at the middle of the cave. This suggests that there may be a fracture pore near the north mouth that allows some air to come in.
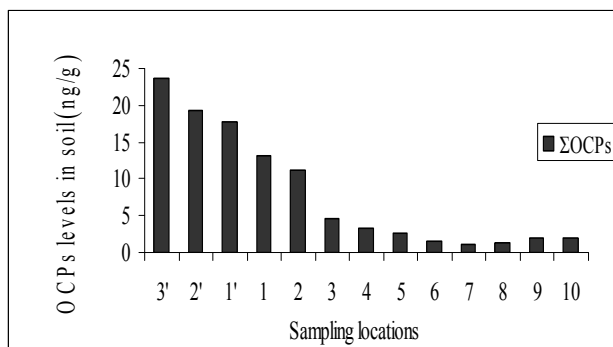
Fig 3: Distribution of ΣOCPs in soil of Dayan cave   Fig 4: Distribution of ΣHCHs and ΣDDTs in soil of Dayan cave
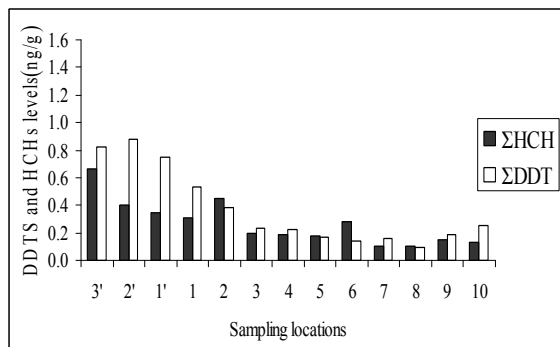
Fig 4 shows that the total concentration DDTs (∑DDTs) in soil samples were higher than the total concentration of HCHs (ΣHCHs). This trend is consistent with the previous observations on the contamination of OCPs in soil in China (Zhou et al., 2001). A most likely explanation for the current low concentrations of HCHs in soil is due to the difference in the physicochemical and biochemical properties, wherein HCHs have higher water solubility, vapor pressure and biodegradability, and lower lipophicility and particle affinity compared to the DDTs (Rui et al., 2005). DDTs tend to remain in the particulate phase longer than HCHs. (Nhan et al., 2001).

In comparison with recent research reports, the concentrations of ∑DDTs and ∑HCHs measured in the study area was in the same low range with other pristine areas such as Tibet plateau where the concentration of ∑DDTs ranged from ND to 2.83 ng/g and ∑HCHs ranged from 0.18 to 5.38 ng/g (Fu et al., 2001), and European high altitude mountains that had ∑DDTs and ∑HCHs residual level in the range of 1.7-13 ng/g and 0.08-0.49 ng/g respectively (Grimalt et al., 2004). The average concentration outside the cave and inside the cave of∑ DDTs and∑ HCHs was lower than the average concentration of ∑DDTs and ∑HCHs which was 0.52 ng/g and 6.19ng/g respectively in Hong Kong soils (Zhang et al, 2006), and they were much lower than the average concentrations of ∑DDTs (37.6 ng/g) and ∑HCHs (12.2 ng/g) found in soils of Pearl River Delta Region (Fu et al., 2003). Some other studies reported around China, had higher residual levels of OCPs such as Beijing (Zhu et al, 2005), Tianjin (Tao et al., 2005), Nanjing (An et al., 2005). In Europe, DDTs and HCHs levels were in the range of 4.3-2400 and 0.36-110 ng/g in Poland soils (Falandysz et al., 2001). In comparison with similar research the levels of OCPs in Guilin were low and the reason is because there are mainly rice farms in the vicinity of Guilin city in which small amounts of OCPs were used with the rotary method of planting rice. The existence of alternating wet and dry conditions is beneficial to the aerobic and anaerobic degradation of OCPs, leading to a reduced amount of soil OCPs.

3.1.1 Distribution and degradation of HCH isomers

It has been widely recognized that HCH is available in two formulations: technical HCH and lindane. Technical HCH contains isomers in the following percentages: α, 55–80%; β, 5–14%; γ, 8–15%; δ, 2–16%; ε, 3–5% (Qiu et al., 2004), and Lindane contains > 90% of γ-HCH. The ratio of α- to γ-HCH has been used to identify the possible HCH source. If the source of HCH comes from fresh input of technical HCH, the ratio of α - to γ -HCH is between 3 and 7 (Yang et al., 2008). However, a lindane source will reduce the ratio to close or <1 (Willet et al., 1998). A higher ratio of α - to γ -HCH than 7 can be explained by long-range transport or re-cycling of technical HCH, because α -HCH has a longer atmospheric lifetime than γ isomer by about 25% (Willet et al., 1998). As shown in Fig 6, the ratios of α-HCH/γ-HCH in all soil sampling sites were lower than 3. Accordingly, the contamination of HCHs in this region probably came from local use of lindane and also indicated new Lindane inputs in the past several years.

By analyzing the individual HCH isomers (Fig 5) we found that β-HCH is the highest isomers detected, among all the samples it accounted from 20.03-79.13 %, especially in sample 3 to 7 where it accounted from 23-79% of the total HCHs detected. The β-HCH was higher because of its persistence in soil. The persistence of β-HCH in soils is mainly due to the higher K_{ow} (log K_{ow} =3.78) and lower vapor pressure value ($3.6x10^{-7}$ mmHg, $20^{o}C$) (Zhang et al., 2006). These will make β-HCH easier to be absorbed to the soil organic matter and less evaporative loss from soils (Mackay et al., 1997). Furthermore, the spatial arrangement of Chlorine atoms in the molecular structure of β-HCH was supposed to be more resistant to microbial degradation in soils (Middeldorp et al., 1996).
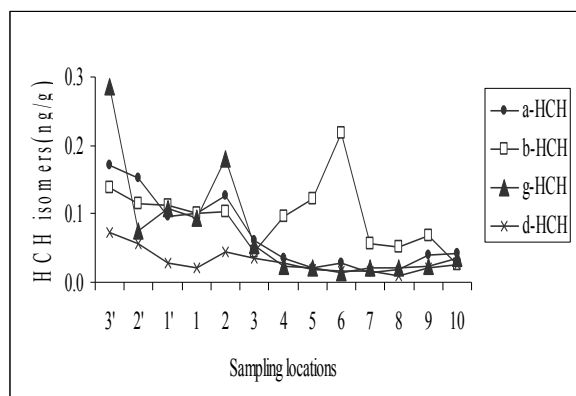
Fig 5: HCH isomers in soil of Dayan cave
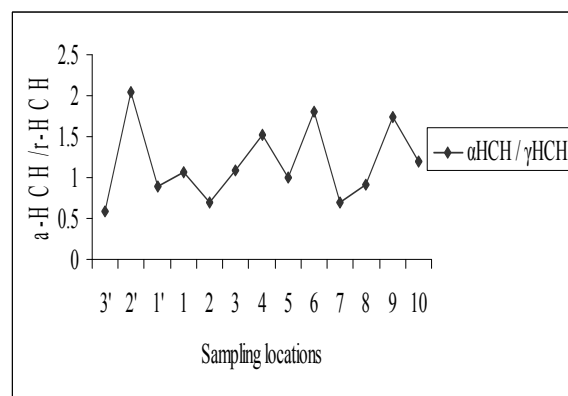
Fig 6: Ratios of α-HCH to γ-HCH in soil of Dayan cave

3.1.2 Distribution and degradation of DDT isomers

Commercial DDT generally contains 75% pp'-DDT, 15% o, p'-DDT, 5%- p, p'-DDE, <0.5% pp'-DDD, <0.5 op'-DDE and <0.5% of unidentified compounds (WHO, 1979), but in Dicofol, the concentration of o, p'-DDT is more than pp'-DDT (Qiu et al., 2005). DDTs isomers have a long persistence in the environment and their levels in this study are shown in Fig. 7.

Since DDT can be biodegraded under aerobic condition to DDE and under anaerobic condition to DDD (Bossi et al., 1992). DDE and DDD Changes in the ratio of DDE and DDD to DDTs has been regarded as an indication of either no or decreasing inputs to the environment. The ratio of (DDE+DDD)/ ΣDDTs >0. 5 can be thought to be subjected to a long term weathering (Dong et al, 2002) and More *o*, *p′*-DDT than *p*, *p′*-DDT in the environment can demonstrate the dicofol-type DDT usage (Qiu et al., 2004).

The ratios of (DDE+DDD)/ ΣDDTs are shown in Fig 8. The ratios were in the range of 0.26-0.61 with most values being less than 0.5 (mean value is 0.4) and in Fig 7 we can see that the concentration of *o, p'* DDT is more than *p, p'* DDT as in Dicofol, this suggests that there is fresh input of Dicofol in the study area. Also, most values of DDD/DDE ratio (Fig 9) were greater than the unity inside the cave, indicating that the soil was dominated by *p, p'*-DDD, the product of anaerobic degradation of *p, p'*DDT (Zhou et al., 2006). DDD/DDE ratios ranged from 0.092 to 7 inside the cave with an average value of 2.31. Contrally outside the cave the ratios of DDD/DDE ranged from 0.052 to 0.53 with an average value of 0.35. The results obtained from the indices clearly indicated that DDTs in soil inside and outside of the Dayan cave may be derived from Dicofol and was retained under anaerobic conditions inside the cave and under aerobic condition outside the cave. The use of Dicofol in China is mainly in the southern and eastern provinces, mostly on litchi, longan, citrus crops and cotton (Yang et al., 2008).
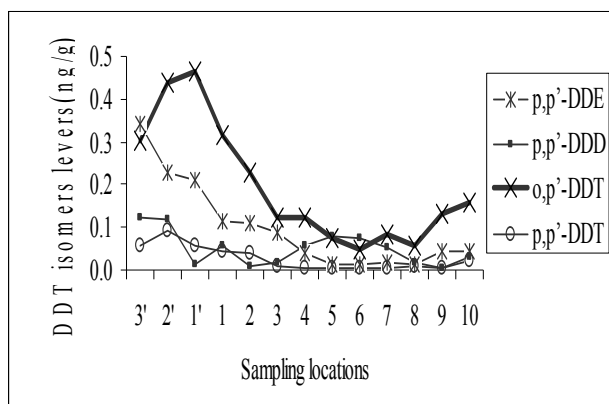
Fig 7 Distribution of DDTs isomers in soil of Dayan Cave
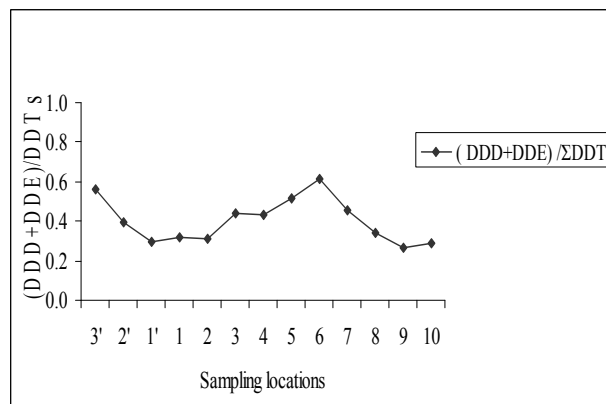
Fig 8: Ratios of (DDD + DDE)/ ΣDDTs in soil of Dayan cave
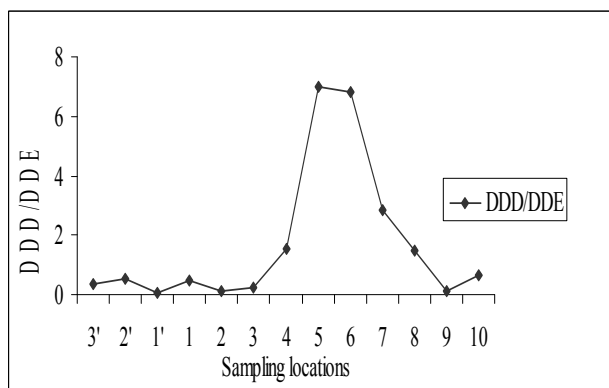
Fig 9 Ratio of DDD/DDE in soil of Dayan Cave

4. Conclusion

The usage of HCHs and DDTs in China has been banned for 20 years and this sanction resulted in a tremendous decrease of OCPs concentrations in soils of Dayan cave. The residual levels of OCPs in soils outside Dayan cave were less than corresponding national values and among all the OCPs detected the concentration of chlordane and heptachlor were highest because they have been largely used in the study area. ΣDDTs and ΣHCHs in soil inside the cave were low in comparison with worldwide background mountains and polar regions. Summarily, the pollution of OCPs in the soils inside and outside Dayan cave was light. The analysis of isomers of DDTs and HCHs showed that there is fresh use of Dicofol and Lindane respectively in the study area. DDTs content in soil outside the cave was mainly aerobically degraded while anaerobic degradation occurred inside the cave.

Acknowledgement

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Fixed Point Theorem And Its Application

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ABSTARCT: This paper examines the existence of fixed point and its application. An introduction to fixed point theorem and its applications to Linear equation are enumerated and proved. Since application usually involved space function, we give the Banach Space of the theorem. [Report and Opinion. 2009;1(1):17-34]. (ISSN: 1553-9873).

Keywords: Fixed point, functions, Banach Space, Non Expansive Map.

INTRODUCTION
Definition 1
The importance of fixed-point theorem in Functional Analysis is due to its usefulness in the theory of ordinary differential equation. The existence of construction of a solution to a differential equation is often reduced to the location of a fixed point for an operator defined on a subset of a space of a function. We use fixed point theorem on many occasions to determine the existence of periodic solution for Functional Differential Equation when solution are already known to exist {1.2}.

Perhaps as a result of the importance of fixed theory in Applied Mathematics and Functional Analysis, it has developed into area of independent research, where several areas of Mathematics such as Homology theory, Degree theory and Differential Geometry have come to play a very significant role

Various attempts have been made by researchers to study and locate existence of solution to a family of Mappings. Thus this study includes the investigation of fixed point theorem for mapping of a set into its power set in I relation to a single-valued mapping {3.4}.

Furthermore, the study of fixed point theorem has developed its own method and ideas as illustrated by Kick {5}

Theorems (1.1)
(1.1) Let (x, d) be a complete metric space. Let $T:x \to 7 X$ be a mapping in the real space. If there exist a number $a<1$ such that $d(T(x), T(y) \leq \alpha d(x,y))$ for each x and y in X_j when the sequence of iterates $\{T^n(X)\}_{n-1}^{\infty}$ converges to point of T for any $X \pounds x$. { 4 }

Remark (1.1)
Remark (1.1): Mapping $T: X \to 7X$ satisfying the condition $d(T(x), T(y) : \leq \alpha d(x,y))$ are called contraction mapping. Theorem (1.1) is involved in many of the existence and uniqueness proofs of Ordinary differential equations.

In order to generalize, the Contraction Mapping theorem to a wider class of function, we have the Brower fixed point theorem stated as follows.

Theorem (1.2): BROWER FLXED POINT THEOREM

Let U be a closed unit ball of any finite dimensional Euclidean spaces. Let $T:U \rightarrow U$ be continuous, then T has a fixed point. Theorem (1.2): is weaker than theorem (1.1) because the sequence of iterates need not converge to a unique fixed point.

An important generalization of theorem (1.2) is the Letschetz Fixed Point theorem which states that given a finite simplicial complex K and a continuous function $T:K \rightarrow k$, it is possible to define a non-negative W(T) called the Lefchetz number of T (with the property that $T \neq 0$) implies the existence of a fixed point of T.

Another important generalization of the Brower Fixed Point theorem is the Schander-Tychoner theorem which is stated as follows:

(1.3)   Let K be a compact subset of a locally convex topological space X. If T is a continuous   mapping from K into k, then T has a fixed point. Attempts at making Theorem (1.3) easier to apply in Functional Analysis leads to the following modification.

(1.4)   Let K be a bounded, closed and convex subset of a Banach space X. Let $T:K \rightarrow k$
be a compact mapping, then T has a fixed point in K.

We need to note that consideration of domains which are only bounded and convex, are example given by Vidossich. Lipschitzian mapping with Lipschitz constant I, may fail to have a fixed point, even under the additional assumption that the domain be compact in the weak-star topology.

This problem was however resolved by Kirk's Theorem which gives further condition ensuring the existence of a fixed point. We thus consider the following definitions before stating Kirks theorem;

Definition (1)

Let X be a Banach space and let $D \subset X$. A mapping D into X is said to be a non-

expansive, if for each x,y ε D, IIT(x)-T(y) II $\leq \|x - y\|$

Definition (2)

Let $K \subset K$, be non convex subset, K is said to have a normal structure, if for each convex subset H of K consist of one point H consists of a non-dimensional point, that is there is a point X_0 in H such that sup

$$\left[\|X_0 - X\| : x \varepsilon H \right] < \sup \left(\|x - y\| : y \varepsilon H \right)$$

Theorem (1.5)

Let X be a Banach space and K a weakly compact convex subset of X, and suppose K has a normal structure, then any non-expensive mapping $T:K \rightarrow K$ has a fixed-point.

Non-expensive mapping have proved to be a great importance in the study of non linear operator, interest in such mappings stems from the fact that they are intimately connected with an important class of operators, the accretive operators, introduced by T. Kato and F.E. Browder in (1967).. Roughly speaking, a mapping T of a normal linear space into itself is accretive if the solution U(t, x) to the initial value problem.

$$\frac{\delta u(t)}{\delta t} + Tu(t) = 0$$
,

where u(0) = x is non expansive in x for each t > 0.

2.1 Definition (3) (Contraction)

Let $X=(x,d)$ be a complete metric space. A mapping $T:X \rightarrow x$ is called a contraction on X if there exist a positive number, $0 < \alpha < 1$, such that $\|Tx - Ty\| = \delta(Tx, Ty) \le \alpha \partial(x, y)$

Definition (4) (Complete metric space

A metric space m is called complete metric space if every Cauchy's sequence converges to a point in M.

2.2 BANACH FIXED POINT THEOREM

Contraction Mapping Principle

Let $X = (x,d)$ be a complete metric space. If $T:X \rightarrow x$ is a contraction, there exist X £ X, such that Tx=x, then T has precisely one fixed point.

Proof: We construct a sequence (X_n) and show that it is Cauchy, so that it converges in the complete space X and then we prove that its limit X is a fixed of T has no fixed points.

We construct a sequence of iterates

$X_{n+1} = 1 + TX_n$

$X_0 : X_1 = TX_0$

$X_1 : X_2 = TX_1$

$X_2 : X_3 = TX_2$ (1.0)

.   .   .

.   .   .

.   .   .

$X_n : \quad X_n = \quad Tx_n$

$\partial (Tx, Ty) \le \alpha \partial (x, y) \quad (a < 1)$ (1.1)

Clearly, the set of equations (1.0) is a sequence of the images of X_o under repeated application of T.

To show that X_n is Cauchy.

By (1.0) and (1.1), we have

$\partial (X_{n+1}, X_n) \quad = \partial (TX_m, TX_{m-1})$

$\alpha \partial (X_m, X_{m-1})$

$0 \le \alpha \partial (TX_m\text{-}, TX_{m-2})$ (1.2)

$\le \alpha^2 \partial (X_m\_l, X_{m-3})$

$\le \alpha^m \partial (X_I, X_0)$

By using triangle inequality which stated that for every $x, y \in X$.

∂ (X$_n$, X$_n$) $\leq \partial$ (X$_m$, X$_{m+1}$) + ∂ (X$_{m+1}$, X$_{m+2}$) +……+ ∂ (X$_{0-1}$, X$_n$)

$\leq \left(\alpha^m + \alpha^{m+1} + ... + \alpha^{n-1}\right)\delta(X_0, X_1)$

By using the formula for the sum of a geometric progression where n>m

$$S_n = \frac{a(1-r)u}{1-r} = a^m \frac{1-a^{u-m}}{1-\alpha}$$

(1.3)

Since 0<α<1 and also $1-\alpha^{n-m}<1$

∂ (X$_m$, Xu) $\leq \dfrac{\alpha^m}{1-\alpha}\delta(X_0, X_1)$, 0<$\alpha$<l.

(1.4)

Therefore, ∂ (X$_o$, X$_1$) is fixed and shows that X$_m$ is Cauchy.

Since X is complete, X$_m$ converges, say X$_m \to$X, as m $\to \infty$

To show that this limit X is a fixed point of the mapping T.

Let, $Tx = x$ and $Ty = y$ (1.5)

$\partial(x, Tx) \leq \partial(x, x_m) + \partial(x_m, Tx) \leq \partial(x, x_m) + \alpha\partial(y_n Ty)$

Then,

Also, $\partial(y, Ty) \leq \partial(y, y_n) + \partial(y_n Ty)$

We conclude that ∂ (x, Tx) = 0, so that x=Tx. These shows that X is a fixed point of T and is the only fixed point of T because from Tx=x and Ty=y, we obtain by (1.2)

∂ (x, y) = ∂ (Tx, Ty) $\to \alpha\partial$ (x, y)

$\Rightarrow \partial$ (x,y) = 0, since $\alpha < 1$

Therefore, x=y and hence the solution is unique.

Theorem (2.3.0) (Contraction on ball)

Let T be a mapping a complete metric space X = (x,d) into itself. Suppose T is a

contraction on closed by Y = $\left\{ x\big/\partial(x, x_0) \leq r \right\}$

That is, T satisfies (1.2) and converges to $X\varepsilon Y$. This X is a fixed point of T and is the only fixed point of T in Y.

Proof: We merely have to show that X$_m$ is as well as X lie in Y. We put m = 0 in (2.2.4),

change n to m. and use equation (1.6) to get $\partial(x_0, x_m) < \dfrac{1}{1-\alpha}\partial(x_0, x_1) < r$

Hence, all X$_m$'s are in Y

Also, $x, y\varepsilon$, since $X_m \to X$ and Y is closed.

2.0   APPLICATION OF FIXED POINT THEOREM

Banach's fixed point theorem has important application to iteration. Three important field of application of the Banach's Linear Space are:

    (i). Linear algebraic equation

    (ii). Ordinary differential equation

    (iii). Integral equation

Here, we restrict ourselves to linear algebraic equation. and consider an

application to Linear equation.

We take the set X of all ordered n-turples of real numbers written as

$$x = (\varepsilon_1, \varepsilon_2, \varepsilon_3,.....\varepsilon n), \ y = (n_1...........n_n), \ z < (\partial_1 / ...., S)$$

On x, we define a metric d by

$$\partial (x, z) = \max / \varepsilon_i - \varepsilon_j / \tag{1.6}$$

$$X = (x, d) \text{ is complete}$$

On X, we define $T:X \rightarrow X$ by $y = Tx = cx + b$ (1.7)

where $C = (C_{jk})$ is a fixed real nxn matrix and $b \varepsilon X$, a fixed vector. In this study, all vectors considered are column vectors, because of the usual conventional of matrix multiplication.

We now have:

$$n_j = \sum_{K=1}^{n} \left(jk^\varepsilon K + \beta j, j = 1,2,...............,n \right.$$

where $b = (\beta i)$

Setting $W = (w_j) = Tz$. We-obtain (1.2) and (1.6)

$$\partial (y,w) = \delta (Tx, Tz) = \max/nj\text{-}wj/$$

$$= \max_j / \sum_{k-1}^{n} cjk \left(^\varepsilon K + ^\infty k \right)/$$

$$\leq \max_j / \varepsilon i - \varepsilon_j / \max_j \sum_{k=1}^{n} / Cjk /$$

$$= \partial(x,z)_j \max \sum_{k=1}^{n} / Cjk /$$

$$\partial(y, w) \leq \alpha \partial(x, z),$$

Therefore,

$$\alpha = \max_j \sum_{k=1}^{n} / Cjk$$

where (1.7)

Theorem (2.5) (Linear Equation)

If a system $x=cx+b$, $c=(Cjk)$, be given as n linear equation in n unknown $\varepsilon_1, \varepsilon_2, \varepsilon_3,.....\varepsilon n$ (the component of x).

$$\sum_{k=1}^{n} / Cjk / < (j = 1,2,.....,n)$$

Satisfies (1.8)

It has precisely one solution x. This solution can be obtained as the limit of the iteration sequence $(x^{(0)}, x^{(1)}, x^{(2)},......)$, where $x^{(0)}$ is arbitrary and

$X^{(m+1)} = cx(m) +$, m = 10. 1 (1.9)

Error bounds are

$$\partial(x^m = x) \leq \frac{\alpha}{1-\alpha} \partial(x^{(m-1)}, x^m) \leq \frac{\alpha^m}{1-\alpha} \partial(x^{(0)}, x^{(1)})$$

(1.10)

Remarks

Condition (1.9) is sufficient for convergence. It is a row sum criteria because it involve row sum obtain by summarizing the absolute values of the elements in row C. if we replaced (1.2)

By other metrics, we would obtain other conditions. A system of non linear equations in n unknown is usually written as Ax = C. where A is an n-rowed square matrix. Many iterative methods for (1.9) with det $A \neq 0$, are such that one writes A=B-G with a suitable non-singular matrix B. then (1.10) becomes Bx=Gx+C or x = $^{e-1}$(G-x+C)

This suggest the iteration (1.9) where, C = B^{-1}G, b=. B^{-1}C. this is illustrated using the following by two standard methods:

i.   The Jacobi, which is largely of theoretical interest.
ii. The Gauss-Seidel iteration, which is largely of use in Applied Mathematics.

(i)   JACOBI ITERATION

This iteration methods is defined as

$$\varepsilon j(m+1) = \frac{1}{ajj}\left(\partial j - \sum_{k=1} ajk\varepsilon_k^{cm}\right), j = 1,2,.................,n.......................(1.4)$$

where C = (vj) in (2.5.3) and we assume ajj $\neq 0$ for j=1,2,............n.

This iteration is suggested by solving the jth equation in (1.4) for ε. It is not difficult to verify that (1.5) can be written as this.

C= -D^{-1}(A-O), b=D^{-I}C.

where D = diag(ajj) is the matrix whose non-zero elements are those of the principal diagonal of A.

Condition (1.4) applied to C in (1.5) is sufficient for the convergence of the Jacobi iteration. Since C in (1.5) is relatively simple, we can express (1.4) directly in terms of the element of A.

The result is the row sum criteria for the Jacobi iteration

$$\sum_{\substack{K=1 \\ K \neq j}}^{n} /\frac{a_{jk}}{a_{jj}}/ \leq 1, j = 1,..n. \tag{1.6}$$

$$or$$

$$\sum_{\substack{K=1 \\ K \neq J}}^{n} / a_{jk} / \leq a_{jj}, j = 1,2,...n \tag{1.7}$$

This shows that convergence is guaranteed if the elements in the principal diagonal of A are sufficiently large.

(ii)   GAUSS-SEIDEL ITERATION

This is a method of successive corrections in which at every instance all the latest known component are used. The method is defined by:

$$\varepsilon j^{(m+1)} = 1\left(Vj - \sum_{k=1}^{j-1} a_{jk}\varepsilon K^{(m+1)} - \sum_{k=j+1}^{n} a_{jk}\varepsilon k^{(m)}\right)$$

$$(1.8)$$

where j=1,2, ... , n and we again assume a$_{jj}$ $\neq 0$ for all j. We obtain a matrix form of (1.8)

above by writing A=-L+D-V
where D is as in the Jacobi iteration and L and *V,* are lower and upper triangular matrix respectively with principal diagonal elements all zero, the minus being a matter of convention and convenience.
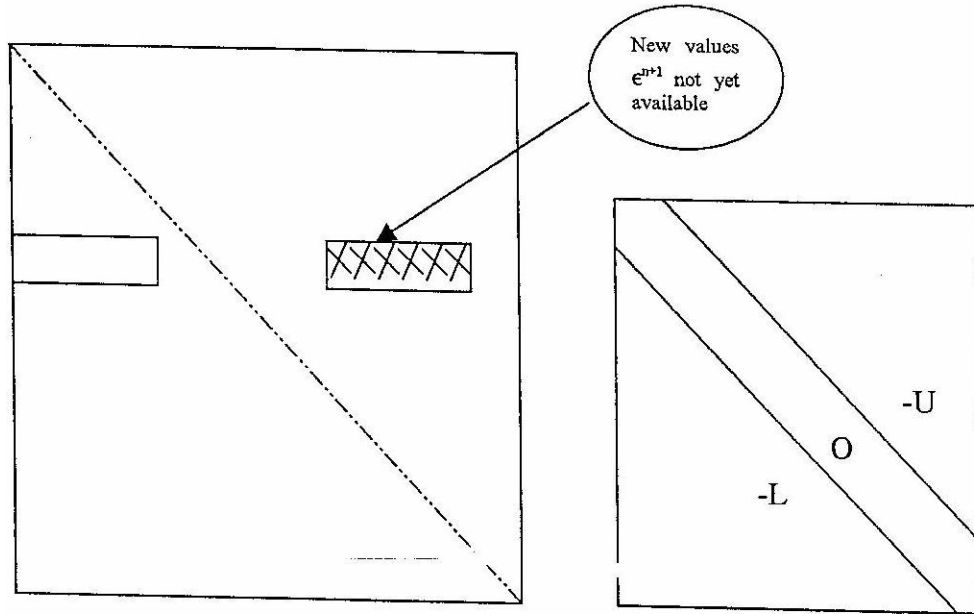
Fig (10) Decomposition of A

Also,

$C = (D-L)^{-1}U$ $b=(D-L)^{-1}C$ (1.9)

Condition 1 applied to C in (1.9) sufficient for the convergence of the Gauss-Seidel iteration. Since C is complicated, the remaining practical problem is to get simpler conditions sufficient for the validity of (1.6).

Example (1.0)

Consider the system

$$z_1 - 0.25z_2 - 0.25z_3 = 0.50$$
$$- 0.25z_1 + z_2 - 0.25z_4 = 0.50$$
$$- 0.25z_1 + z_2 - 0.25z_4 = C$$
$$- 0.25z_1 + g_3 - 0.25z_4 = 0.25$$
$$- 0.25z_2 - 0.25z_3 + z_4 = 0.25$$

(a). Equations of these form arise in the numerical solution of partial differential equation.

(ii) Apply the Jacobi iteration, starting from X(0) with components (1, 1 , 1 , 1) the performing three stages.

Compare the approximating value with the exact values

$z_1 = z_2 = 0.875$, $z_3 = z_4 = 0.625$

(b). Apply the Gauss-Seidel iteration, performing the same tasks as in (a)

SOLUTION TECHNIQUE

$$\begin{bmatrix} 100 & -25 & -25 & 0 \\ -25 & 100 & 0 & -25 \\ -25 & -25 & -25 & 100 \\ 0 & 25 & -25 & 100 \end{bmatrix} \begin{bmatrix} z_1 \\ z_2 \\ z_3 \\ z_4 \end{bmatrix} = \begin{vmatrix} 50 \\ 50 \\ 25 \\ 25 \end{vmatrix}$$

$$Ax_j = B_j$$

$$x(0) = \begin{bmatrix} z_1(0) = 1 \\ z_2(0) = 1 \\ z_3(0) = 1 \\ z_4(0) = 1 \end{bmatrix}$$

(a) Using the formular $z_j (m + 1) = \dfrac{1}{a_{jj}} \left(v_j - \sum\limits_{\substack{k=1 \\ k \neq 1}}^{n} \partial jk - \varepsilon_k^{(m)} \right)$

where V_j =diagonal elements, m=0, j=1

$$z_1^{(1)} = \frac{1}{a_{11}} \left(\partial_1 - \sum_{k=2}^{n} a_{1k} \varepsilon_k^{(0)} \right)$$

$$= \frac{1}{100}(50+50)$$

$$= 1.0000$$

When J=2, m=0

$$z_2(1) = \frac{1}{a_{22}} \left(v_j - \sum_{\substack{k=1 \\ k \neq 2}}^{n} \partial jk - \varepsilon_k^{(m)} \right)$$

$$= \frac{1}{100}(50+50)$$

$$= 1.0000$$

When m=0,J=3

$$z_3(1) = \frac{1}{a_{33}} \left(v_j - \sum_{\substack{k=1 \\ k \neq 3}}^{n} \partial jk - \varepsilon_k^{(m)} \right)$$

$$= \frac{1}{100}(50+25)$$

$$= 0.7500$$

When m=0,J=4

$$z_4(1) = \frac{1}{a_{44}}\left(v_j - \sum_{\substack{k=1 \\ k \neq 4}}^{4} \partial jk - \varepsilon_k^{(0)}\right)$$

$$= \frac{1}{100}\ (50+25)$$

$$= 0.7500$$

$$X^{(1)} = \begin{bmatrix} x_1^{(1)} \\ x_2^{(1)} \\ x_3^{(1)} \\ x_4^{(1)} \end{bmatrix} = \begin{pmatrix} 1.0000 \\ 1.0000 \\ 0.7500 \\ 0.7500 \end{pmatrix}$$

From m=1, j =1
Using the fomular

$$z_1^{(2)} = \frac{1}{a_{jj}}\left(\gamma_j - \sum_{\substack{k=1 \\ k \neq j}}^{4} a_{jk}\varepsilon_k^{(1)}\right)$$

$$z_1^{(2)} = \frac{1}{a_{11}}\left(\partial_j - \sum_{\substack{k=1 \\ k \neq 4}}^{4} a_{1k}\varepsilon_k^{(0)}\right)$$

$$= \frac{1}{100}\left(50 - \left(a_{12}\varepsilon_1^{(1)} + a_{13}\varepsilon_3^{(1)}\right)\right)$$

$$= \frac{1}{100}\left(50 - \left(-25 \times 1 + \left(-25 \times 0.75\right)\right)\right)$$

$$= 0.9375$$

Using similar approach
Where m=1, J=2

$$z_2^{(2)} = \frac{1}{a_{22}}\left(\partial_2 - \sum_{\substack{k=1 \\ k \neq j}}^{4} a_{2k}\varepsilon_k^{(1)}\right)$$

$$= 0.9375$$

In the same way,
When m=1, J=3

$$z_3^{(2)} = \frac{1}{a_{33}}\left(\partial_3 - \sum_{\substack{k=1 \\ k \neq 3}}^{4} a_{3k}\varepsilon_k^{(1)}\right)$$

$$= 0.6875$$

Also, when m=1, J=4

$$z_4^{(2)} = \frac{1}{a_{44}} \left(\partial_4 - \sum_{\substack{k=1 \\ k \neq 4}}^{4} a_{4k} \varepsilon_k^{(1)} \right)$$

$$= \frac{1}{100} \left(25 + \frac{175}{4} \right)$$
$$= 0.6875$$

Therefore,

$$x^{(2)} = \begin{bmatrix} z_1^{(2)} \\ z_2^{(2)} \\ z_3^{(2)} \\ z_4^{(2)} \end{bmatrix} = \begin{pmatrix} 0.9375 \\ 0.9375 \\ 0.6875 \\ 0.6875 \end{pmatrix}$$

When m=2, J=1
Using the following formula

$$Zj^{(m+1)} = \frac{1}{a_{jj}} \left(Yj - \sum_{\substack{k=1 \\ k \neq j}}^{4} a_{jk} \varepsilon_k^{(1)} \right)$$

$$z_1^{(3)} = \frac{1}{a_{11}} \left(\partial_4 - \sum_{\substack{k=1 \\ k \neq 1}}^{4} a_{1k} \varepsilon_k^{(2)} \right)$$

$$= \frac{90.625}{100}$$

When m=2, J=2

$$z_2^{(3)} = \frac{1}{a_{22}} \left(\partial_2 - \sum_{\substack{k=1 \\ k \neq 2}}^{4} a_{2k} \varepsilon_k^{(2)} \right)$$

$$= \frac{1}{100} \left(50 - \left(-23.4375 + (-17.1875) \right) \right)$$

$$= \frac{1}{100} \left(50 + 40.625 \right)$$

$$= \frac{90.625}{100}$$

$$= 0.90625$$

$$z_3^{(3)} = \frac{1}{a_{33}} \left(\partial_3 - \sum_{\substack{k=1 \\ k \neq 3}}^{4} a_{3k} \varepsilon_k^{(2)} \right)$$

$$= \frac{1}{100} \left(25 - \left(-25 \times 0.9375 \right) + 0 + \left(-25 \times 0.6875 \right) \right)$$

$$= 0.65625$$

When m=2, J=4

$$z_4^{(3)} = \frac{1}{a_{44}} \left(\partial_4 - \sum_{\substack{k=1 \\ k \neq 4}}^{4} a_{4k} \varepsilon_k^{(2)} \right)$$

$$= \frac{1}{100} \left(25 - \left(a_{41} g_1^{(2)} + a_{42} g_2^{(2)} + a_{43} g_3^{(2)} \right) \right)$$

$$= \frac{1}{100} \left\{ 25 - \left(0 + \left(-25 \times 0.9375 \right) + \left(-25 \times 0.6875 \right) \right) \right\}$$

$$= 0.65625$$

Therefore,

$$X^{(3)} = \begin{bmatrix} z_1^{(3)} \\ z_2^{(3)} \\ z_3^{(3)} \\ z_4^{(3)} \end{bmatrix} = \begin{pmatrix} 0.90625 \\ 0.90625 \\ 0.65625 \\ 0.65625 \end{pmatrix}$$

To compare the approximation with the exact value $g_1 = g_2 = 0.825, g_3 = g_4 = 0.625$
Actual Error = Approximate-exact value

$$= 0.90625\text{-}0.875$$
$$= 0.03125 \text{ (for } z_1 = z_2)$$

Actual error for ($g_3 = g_4$)
= Approximate value-exact value

$$= 0.65625\text{-}0.625$$
$$= 0.03125$$

But X=

$$\begin{bmatrix} 0.875 \\ 0.625 \end{bmatrix}$$

The maximum value is 0.3125<1

The actual error is 0.3125, which indicates that the formula is still good for stability and convergence. To calculate error bound we have

$$\partial\left(X^{(3)}, X\right) = / X^{(3)} - X / \leq \frac{\alpha}{1-\alpha} \left(X^{(2)}, X^{(3)}\right)$$

$$\leq \frac{\alpha^m}{1-\alpha} \partial\left(X^{(0)} X^{(1)}\right) \qquad m = 3$$

$$= \frac{\alpha^3}{1-\alpha} \partial\left(X^{(0)}, X^{(1)}\right)_.$$

We pick largest value from $X^{(3)}$
i.e. $\alpha = 0.5$ from convergence criteria

$$\partial\left(X^{(0)}, X^{(1)}\right) = d\begin{bmatrix} 1-1 \\ 1-1 \\ 1-0.75 \\ 1-0.75 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0.25 \\ 0.25 \end{bmatrix}$$

But,

But 0.25 is the maximum.

Error bound now is

$$\partial\left(X^{(3)}, X\right) = \frac{(0.5)^3 \cdot 0.25}{1-0.5} = \frac{0.031625}{0.5} = \frac{0.031625}{0.5}$$
$$= 0.0625$$

Which is the same as the first method

Actual error $\qquad = \qquad |\text{Approximate - Exact}|$

$= 0.90625\text{-}0.875 \qquad\qquad = 0.03125$

$= 0.65625\text{-}0.625 \qquad\qquad = 0.03125$

Which shows that the formular is also accurate for the stability and convergence of the solution.

(b) Using the Formular,

$$zj^{(m+1)} = \frac{1}{a_{jj}}\left(Vj = \sum_{k=1}^{j-1} a_{jk}\varepsilon_k^{(m)} - \sum_{k=j+1}^{n} a_{jk}\varepsilon_k^{(m)}\right)$$
$$= \frac{1}{100}(50 + 50)$$
$$= 1.0000$$

When m=0, J=2

$$Z_2^{(1)} = \frac{1}{a_{22}}\left(\partial_2 - \sum_{k=1}^{1} a_{2k}\varepsilon_K^{(1)} - \sum_{k=3}^{4} a_{2k}\varepsilon_k^{(0)}\right)$$
$$= \frac{1}{100}\left(50 + 25g_1^{1} + 25\right)$$
$$= \frac{1}{100}(50 + 50)$$

$$= 1.0000$$

When m=0, J=3

$$Z_3^{(1)} = \frac{1}{a_{33}}\left(\partial_3 - \sum_{k=1}^{2} a_{3k}\varepsilon_K^{(1)} - \sum_{k=4}^{4} a_{3k}\varepsilon_k^{(0)}\right)$$
$$= \frac{1}{100}\left(25 + 25_1^{1} + 25\right)$$
$$= \frac{75}{100} \qquad\qquad = 0.7500$$

When m=0, J=4

$$Z_4^{(1)} = \frac{1}{a_{44}}\left(\partial_4 - \sum_{k=1}^{2} a_{4k}\varepsilon_K^{(1)} - \sum_{k=5}^{4} a_{4k}\varepsilon_k^{(0)}\right)$$

$$= \frac{1}{100}\left(25 - \left[a_{4k}g_1^{(1)} + a_{42}g_2^{(1)} + a_{43}g_3^{(1)}\right]\right)$$

$$= \frac{1}{100}\left(25 - \left(0 + \left(25 \times 1 + \left(-25 \times 0.7500\right)\right)\right\}\right.$$

$$= \frac{1}{100}\left(25 + 4375\right)$$

$$= 0.6875$$

$$X^{(3)} = \begin{bmatrix} g_1^{(1)} \\ g_2^{(1)} \\ g_3^{(1)} \\ g_4^{(1)} \end{bmatrix} = \begin{pmatrix} 1.0000 \\ 1.0000 \\ 0.7500 \\ 0.6875 \end{pmatrix}$$

For m=1, J=1

$$Z_1^{(2)} = \frac{1}{a_{11}}\left(\partial_1 - \sum_{k=1}^{0} a_{1k}\varepsilon_K^{(2)} - \sum_{k=2}^{4} a_{1k}\varepsilon_k^{(1)}\right)$$

$$= \frac{1}{100}\left(50 + 25 + 18.75\right)$$

$$= \frac{1}{100}\left(93.75\right)$$

$$0.9375$$

$$Z_2^{(2)} = \frac{1}{100}\left(50 - \sum_{\substack{k=1 \\ 1}}^{0} a_{2k}\varepsilon_K^{(2)} - \sum_{\substack{k=3 \\ 4}}^{4} a_{2k}\varepsilon_k^{(1)}\right)$$

$$= \frac{1}{100}\left(90.63\right)$$

$$= 0.9063$$

When m=1, J=3

$$z_3^{(2)} = \frac{1}{100}\left(25 - \sum_{k=1}^{2} a_{3k}\varepsilon_K^{(2)} - \sum_{k=4}^{4} a_{3k}\varepsilon_k^{(1)}\right)$$

$$= \frac{1}{100}\left(65.63\right)$$

$$= 0.6563$$

When m=1, J=4

$$z_4^{(2)} = \frac{1}{100}\left(25 - \sum_{k=1}^{3} a_{4k}\varepsilon_K^{(2)} - \sum_{k=5}^{4} a_{4k}\varepsilon_k^{(1)}\right)$$

$$z_4^{(2)} = \frac{1}{100}\left(25 - \sum_{k=1}^{3} a_{4k}\varepsilon_K^{(2)}\right)$$

$$= \frac{1}{100}(64.07)$$

$$= 64.07/100 \qquad = 0.6407$$

$$X^{(2)} = \begin{bmatrix} z_1^{(2)} \\ z_2^{(2)} \\ z_3^{(2)} \\ z_4^{(2)} \end{bmatrix} = \begin{pmatrix} 0.9375 \\ 0.9063 \\ 0.6563 \\ 0.6407 \end{pmatrix}$$

When m=2, J=1

$$z_j^{(3)} = \frac{1}{a_{jj}}\left(\partial_j - \sum_{k=1}^{j-1} a_{jk}\varepsilon_K^{(3)} - \sum_{k=j+1}^{n} a_{jk}\varepsilon_k^{(2)} \right)$$

$$z_j^{(3)} = \frac{1}{100}\left(50 - \sum_{k=1}^{0} a_{01}z_1^{(3)} + a_{13}z_3^{(2)} + a_{14}z_4^{(2)} \right)$$

$$= \frac{1}{100}\left(50 - -25 \times 0.9063 - 25 \times 0.6563 + 0 \right)$$

$$= \frac{1}{100}\left(50 + 39.065 \right)$$

$$= \frac{1}{100}(89.065)$$

$$= 0.89065$$

When m=2, J=2

$$z_j^{(3)} = \frac{1}{a_{22}}\left(V_2 - \sum_{k=1}^{1} a_{21}\varepsilon_K^{(3)} - \sum_{k=3}^{n} a_2\varepsilon_k^{(2)} \right)$$

$$z_2^{(3)} = \frac{1}{100}\left(50 - \sum_{k=1}^{0} a_{21}z_1^{(3)} + a_{23}Z_3^{(2)} + a_{24}z_4^{(2)} \right)$$

$$= \frac{1}{100}\left(50 - \left(-25 \times 0.8907 + 25 \times 0.6407 \right) \right)$$

$$= \frac{1}{100}\left(50 + 38.285 \right)$$

$$= \frac{88.285}{100}$$

$$= 0.8829$$

When m=2, J=3

$$Z_3^{(3)} = \frac{1}{a_{33}}\left(V_3 - \sum_{k=1}^{2} a_3\varepsilon_K^{(3)} - \sum_{k=4}^{n} a_3\varepsilon_k^{(2)} \right)$$

$$= \frac{1}{100}\left(25 - \left(-25 \times 0.8907 + 25 \times 0.6407 \right) \right)$$

$$= \frac{1}{100}\left(25 - \left(-38.285 \right) \right)$$

$$= \frac{63.285}{100}$$

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$$= 0.6329$$

When m=2, J=4

$$Z_4^{(3)} = \frac{1}{a_{44}}\left(V_4 - \sum_{k=1}^{3} a_4 \varepsilon_K^{(3)} - \sum_{k=5}^{n} a_4 \varepsilon_k^{(2)}\right)$$

$$= \frac{1}{100}\left(25 - a_{41} z_1^{(3)} + a_{42} z_2^{(2)} + a_{43} z_3^{(2)} + 0\right)$$

$$= \frac{1}{100}\left(25 - \left(0 - 25 \times 0.8829 - 25 \times 0.6329\right)\right)$$

$$= \frac{1}{100}\left(25 - \left(-37.895\right)\right)$$

$$= \frac{62.895}{100}$$

$$= 0.62895$$

$$X^{(3)} = \begin{bmatrix} z_1^{(3)} \\ z_2^{(3)} \\ z_3^{(3)} \\ z_4^{(3)} \end{bmatrix} = \begin{pmatrix} 0.8907 \\ 0.8829 \\ 0.6329 \\ 0.6290 \end{pmatrix}$$

Example (1.1)

Consider the system

$$5z_1 - z_2 = 7$$
$$-3z_1 + 10z_2 = 24$$

(a) Determine the Jacob iteration. Does C satisfies function starting with $X^{(0)} = 1, x_2(0) = 2$. Calculate $x^{(1)}$, $x^{(2)}$ and the error bounds for $x^{(2)}$.

Solution:

$$A = \begin{bmatrix} 5, & -1 \\ -3 & 10 \end{bmatrix}, \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} = \begin{bmatrix} 7 \\ 24 \end{bmatrix}$$

$$Ax = d$$

$$\sum_{k=1}^{n} / \frac{ajk}{ajj} / < \frac{3}{5} < 1, \frac{1}{10} < 1$$

$$X^{(0)} = \begin{bmatrix} 1, z_1^{(0)} \\ 1, z_2^{(0)} \end{bmatrix}$$

Using the Formular

$$= \quad zj^{(m+1)} = \frac{1}{ajj}\left(\dot{\jmath} - \sum_{k=1}^{n} a_{jk} \varepsilon_k\right)$$

When m=0, J=1

$$z_1^{(1)} = \frac{1}{a_{11}}\left(\partial_1 - \sum_{k=1}^{2} a_{1k} \varepsilon_k^{(0)}\right)$$

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$$= \frac{1}{5}\left(7 - (-1) \times 1\right)$$

$$= \frac{8}{5}$$

When m=0, j=2

$$z_2^{(0)} = \frac{1}{a_{22}}\left(\partial_2 - \sum_{k=1}^{2} - \sum_{\substack{k=1 \\ k \neq 1}}^{2} a_{2k}\varepsilon_k^{(0)}\right)$$

$$= \frac{1}{10}\left(24 - (-3) \times 1\right)$$

$$= \frac{27}{10}$$

$$X^{(1)} = \begin{bmatrix} \frac{8}{5} \\ \frac{27}{10} \end{bmatrix}$$

When m=a, J=1

$$z_2^{(2)} = \frac{1}{a_{22}}\left(\partial_2 - \sum_{k=1}^{2} - \sum_{\substack{k=1 \\ k \neq 1}}^{2} a_{2k}\varepsilon_k^{(1)}\right)$$

$$= \frac{1}{10}\left(24 + 3 \times \frac{8}{5}\right)$$

$$= \frac{144}{50} \qquad\qquad = \frac{70}{25}$$

$$X^{(2)} = \begin{bmatrix} \frac{97}{50} \\ \frac{72}{25} \end{bmatrix}$$

To calculate the error bound

$$\partial\left(x^{(2)}, x\right) = / x^{(2)} - x / < \frac{\alpha \partial}{1-\alpha}\left(x^{(1)}, x^{(1)}\right)$$

$$\leq \frac{\alpha^2}{1-\alpha}\partial\left(x^{(0)}, x^{(1)}\right)$$

$$\alpha = \frac{3}{5}$$

From convergence criteria

$$\partial\left(x^{(2)}, x\right) \leq \frac{3/5}{1-3/5}\partial\left(x^{(2)}, x^{(2)}\right) \leq \frac{3/5}{1-3/5}\partial\left(x^{(0)}, x^{(1)}\right)$$

$$\max_{j} / x_i - y_i /$$

Now, $\partial\left(x^{(0)}, x^{(1)}\right) = \max\left(\frac{3}{5}, \frac{17}{10}\right)$

Now,
$$\partial\left(x^{(2)},x\right)\le\frac{\left(3/15\right)^2}{1-\left(3/5\right)}$$

Error bounds

$$X^2-X=\begin{bmatrix}97/50 & -2\\72/25 & -3\end{bmatrix}=\begin{pmatrix}3/50\\-3/25\end{pmatrix}$$

NB: Absolute choice of higher value is $\left(3/25\right)$

When m=0,j=3

$$Z_3^{(1)}=\frac{1}{a_{33}}\left(\partial_3-\sum_{\substack{k=1\\k\ne3}}^{3}a_{3k}\,\varepsilon_k^{(0)}\right)$$

$$=\frac{1}{100}\left(25+50\right)$$

$$=\frac{75}{100}=0.7500$$

When m=J=4

$$Z_4^{(1)}=\frac{1}{a_{44}}\left(\partial_4-\sum_{\substack{k=1\\k\ne4}}^{3}a_{4k}\,\varepsilon_k^{(0)}\right)$$

$$\frac{1}{100}\left(25+50\right)$$

$$=\frac{75}{100}=0.7500$$

$$X^{(1)}=\begin{bmatrix}z_1^{(0)}\\z_2^{(0)}\\z_3^{(0)}\\z_4^{(0)}\end{bmatrix}=\begin{pmatrix}1.0000\\1.0000\\0.7500\\0.7500\end{pmatrix}$$

NON-EXPANSIVE MAPPING

(3.0)

Recall from the definition above that if x is a Banach space and DCX, then a mapping D into X is said to be non-expansive if for each x,y ε D //Tx-Ty// $\le$ //x-y//.

Consider the iterative Scheme $X_{n+1}=Tx^n$, $x_0\varepsilon X$ ....(3.0)

If T is non-expensive, that is if //Tx-Ty// $\le$ //x-y// are there exist $X^*\varepsilon K,KCX$, such that $TX^*=X^*$.

Can we approximate X^* by a sequence of iterates of T?

The answer is NO

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Reason

Consider
$$T\begin{Bmatrix} X_1 \\ X_2 \end{Bmatrix} = \frac{1}{\sqrt{2}}\begin{bmatrix} 1 & -1 \\ 1 & -1 \end{bmatrix}\begin{bmatrix} X_1 \\ X_2 \end{bmatrix}$$

Let A= (aij) such that

$$//A// = \underset{n}{Max}\left\{\sum_{j=1}^{n} / aij /^2 \right)^{\frac{1}{2}}\right\}..................... *$$

* Is a norm, T is Non-expensive

To see this

$$//Tx\text{-}Ty// = //\frac{1}{\sqrt{2}}\begin{bmatrix} 1 & -1 \\ 1 & -1 \end{bmatrix}^{(x,y)} //$$

$$= \frac{1}{\sqrt{2}}\begin{bmatrix} 1 & -1 \\ 1 & -1 \end{bmatrix}//^{(x,y)}//$$

Therefore, $//Tx\text{-}Ty// \leq //x\text{-}y//$ which shows that T is Non-expansive.

Now, Tx=x………………………..(3.1)

$$As = \frac{1}{\sqrt{2}}\begin{bmatrix} 1, & -1 \\ 1, & 1 \end{bmatrix}^{(x)}$$

From (3.0.1)

Tx-X=0

It implies that $= \frac{1}{\sqrt{2}}\left\{\begin{bmatrix} 1, & -1 \\ 1, & 1 \end{bmatrix} - \begin{bmatrix} 1, & 0 \\ 0, & 1 \end{bmatrix}\right\}x = 0........................(3.2)$

Solving (3.2) implies x=0

This implies that the iterates cannot converse.

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Some Studies on Filariasis and associated biochemical alteration in Egyptian buffaloes

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Abstract

Filariasis are vector-transmitted parasites, exclusively tropical, except for dirofilariosis, it is associated with a heavy burden of morbidity and mortality in ruminants. This investigation was carried on one hundred Egyptian buffaloes, 1.5 - 5 years, from different private farms located in Kaliobia province in Egypt, suffering from fever (40-41ºC), anorexia, enlargement of superfacial lymph nodes, cutaneous sub cutaneous and intramuscular connective tissues ulcers and skin necrosis causing a seasonal haemorhagic dermatitis on skin. Laboratory examination indicated the presence of filaria in the blood. The serum investigations using some biochemical parameters measurments revealed some increase in lipid peroxidation product serum Malondialdehyd (MDA) and a decrease in serum total protein and albumin in diseased animals. Protein electrophoretic patterns showed declined values of α_2a ,α_2b, Total α, β_1, and γ_1 and total glubins concentrations in contrast $\gamma2$ fractions.
 Mass drug administration study was reported here when infected animals were treated with two doses of Ivermectin (1 ml/50kg b.w) in combination to levamesol (1 ml/20 kg b.w) with two weeks interval, a marked recovery was noticed and confirmed by the serum biochemical analysis of treated animals. [Report and Opinion. 2009;1(1):35-44]. (ISSN: 1553-9873).

Key wards: Filariasis, Buffalo, Biochemical analysis, Disease control.

Introduction

Filariasis is widespread throughout the developing world and are associated with a heavy burden of morbidity and mortality resulting in considerable economic losses each year to beef and dairy buffaloes.

Filariasis is debilitating parasitic disease in many Tropical Countries. Despitethe highly evolved immune system, The Filarial parasites Successfully evade host immunity to persist for sustained period of time. Filarial parasites achieve this long – Term Survival Through release of immunosuppressive materials in host. (Muthian *et al*, 2006).

Filariasis is vector-transmitted parasites, transmitted by arthropod vectors, they are endemic in tropical and subtropical regions of the world, their impact differs according to the type of filaria and the induced immune response.

Diagnosis of filaria is made based on the presence of dermatological or lymphatic manifestations, it can also be established following a laboratory examination revealing adverse effects on some biochemical parameters or correspond to the

incidental finding of microfilariae (blood or skin), the visualization of the adult parasite confirms the infection. (souls by, 1968)

The specific laboratory diagnosis of filariasis depends either on the demonstration of circulating microfilaria in the peripheral blood or various stages of the parasite in tissue sections. Various morphological characteristics of the parasite will normally assist in its identification. Concentration techniques, especially those using the polycarbonate membrane filtration of a ml or more of heparinised blood, can detect the parasite in those with very low microfilaria counts (Mak , 2004). Filariasis symptoms are usually caused by the adult worms, where microfilariae cause slight pathological alteration. The presence of the adult worm in lymphatics gives rise to inflammatory lesions and fibrosis which may result in some obstructions of lymphatics, overgrowth of fibrous tissues around dead worms and lymphatic vessels may also rupture this in association with different biochemical functions changes.

The present report deals with alterations in malonalhehyde (MDA) and total protein Fraction in blood of *filariasis infected* buffaoes before and after treatment.

There is little doubt that combination therapy mass drug administration represents a significant advance in treatment of filariasis (Maged el setouhy *et al*., 2004)**,** numerous field trials evaluating the efficacy in the control of lymphatic filariasis have been conducted,

In this study, three trends of investigations were utilized, first is the clinical examination and diagnosis, second is the biochemical alteration in the infected and treated animals, concerning the MDA, serum protein and its electrophoretic pattern was performed, third concerns with the efficacy of utility of two drugs in particular, ivermectin (IVM) and Levamesole for effectiveness in the treatment of lymphatic filariasis.

Material and Methods

Animals
One hundred buffaloes 1.5-5 years from different private farms located in kaliobia were clinically examined.

Samples
Venous blood samples were collected on anticoagulant for direct smear parasitological examination. and other blood Sample without anticoagulant were collected at clean plastic centrifuge tubes and allowed to coagulate , The Serum were separated by centrifugation at 300 r.pm for 10 minute then clear supernatant serum aspirated carefully into dry and sterile labeled vials and used for serum analysis.

Microscopic examination
Identification of microfilariae by microscopic examination is the most practical procedure. Examination of blood samples will allow identification of adult filaria and microfilariae. It is important to time the blood collection with the known periodicity of the microfilariae. Venous blood samples were collected on anticoagulant before and after the treatment for direct smear parasitological examination . The detection of microfilaria was made by direct smear (the simplest method described by (Soulsby, E.J.L.1968) as follow:

1- Place one drop of venous blood onto a clean microscope slide.
2- Place a coverslip over the drop of blood.
3- Examine the coverslip area under low magnification (X 100) of microscope, look fore the undulating of larvae.

Serum analysis

Blood samples without anticoagulant for serum analysis during infection and 4 weeks after treatment were collected.

Serum was used for determination of total protein and its electrophoretic pattern, this was carried out according to *SonnenWirth and Jaret (1980); Davis (1964) and Ornstein (1964) respectively.* Also, MDA was measured by thiobarbituric acid method, a modified form of the procedure described by *(Ohkawa 1979).*

Treatment

Infected animals were treated with two doses, Ivermictin manufactured by Arab Company for Medical Peoducts), S/C injection in a dose of (1 mg/50Kg body weight in addition to Levamesole 7.5% manufactured by El. Nasr pharmaceutical chemicals co . Hydrochloride S/C injection in a dose of (1 mg/ 20Kg body weight) with 2 weeks intervals for the two drugs

Statistical analysis:

Data obtained were statistically computed for ANOVA   test and using least significant difference (LSD)   for comparison between means at $p < 0.05$, using SPSS 14 (2006).

Results

Clinical signs of infected animals

The clinical investigation of infected animals revealed that the different infected buffaloes were suffering fever (40-41ºC), anorexia, enlargement of superficial lymph nodes, cutaneous subcutaneous and intramuscular connective tissues ulcers, nodules and skin necrosis causing a seasonal haemorhagic dermatitis on skin (filarial dermatitis), and abcesses in some cases. The nodules open spontaneously and produced a haemorrhagic exudates.(Souls by , 1968)
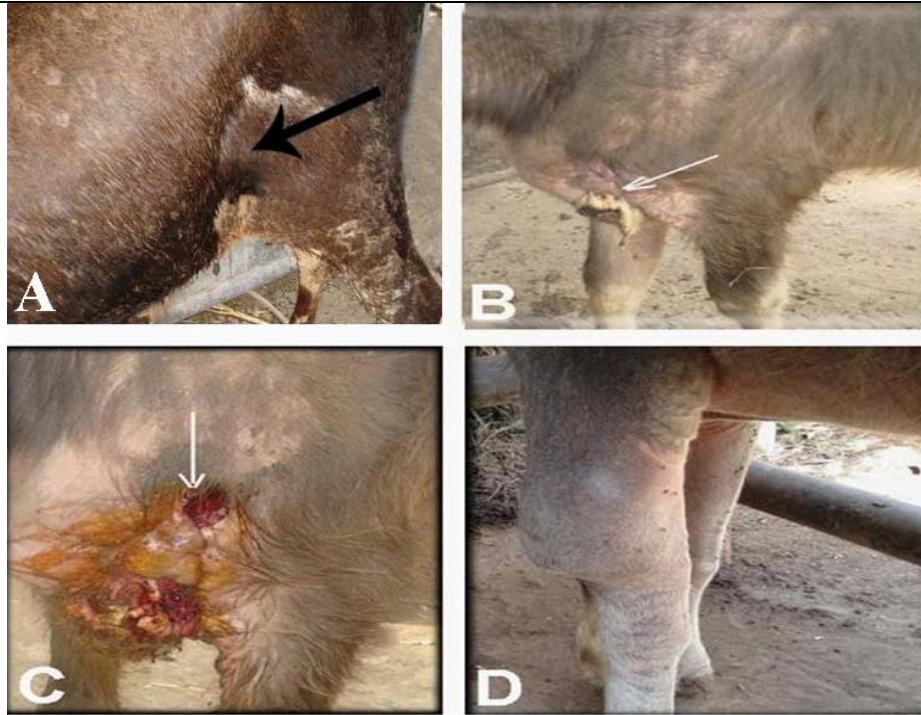
Figure 1: Different lesions of filariasis;

A, superficial lymph node enlargement.

B, spontaneously opened nodule.

C, surgical incision.

D, knee abcess and oedma.

Microscopic examination

Microscopic examination before treatment confirmed the presence of the adult and the microfilaria in the infected animal's blood, the microscopic examination of the same animals after treatment confirmed the disappearance of the filarial parasites in the blood.

Figure 2: An impression smear of the filaria and the RBC in the capillaries
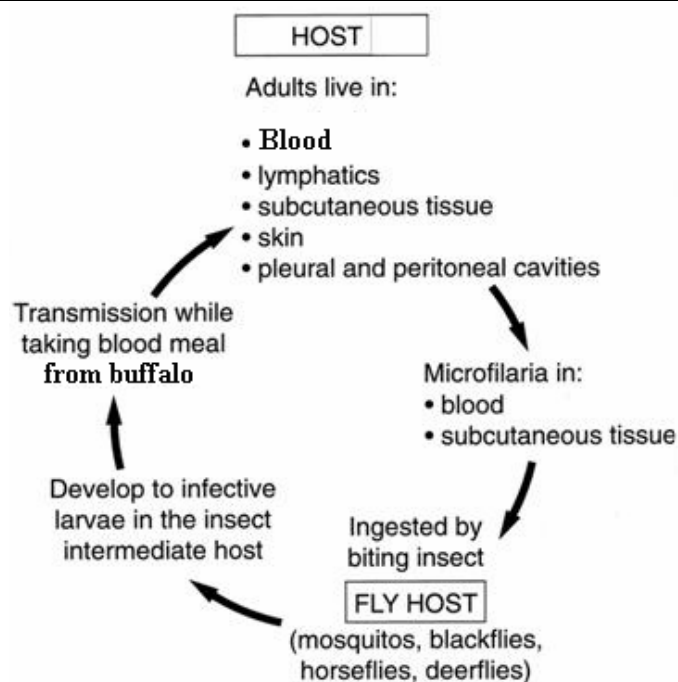
Figure 3: Basic life cycle of filarial

Biochemical Analysis

From gel electrophoresis (Fig. 3) and from the statistical analysis examplified in table 1 , It is evidenced that, diseased buffaloes with filaria reported hypoproteinemia hypoalbuminemia , hypoglboblinemia (α,2a ; α,2b β1 and γ1,) and reduced A/G ratio was significantly increased as noticed after treatment.There was a significant drop in albumin value and consequently total protein.

Table (1): Total serum protein and its electrophoretic patterns   concentrations (gm/dl) in control, diseased and   treated buffaloes.

Parameter	Control	Diseased	Treated	LSD
T.P	8.478±0.043 [a]	6.724±0.1745 [c]	8.028±0.1026 [b]	0.3683***
Albumin	3.427±0.018 [b]	2.439±0.039 [c]	3.817±0.199 [a]	0.363***
α_1	0.280±0.005 [b]	0.250±0.016 [b]	0.437±0.014 [a]	0.0386***
α_2a	0.413±0.004 [a]	0.282±0.007 [b]	0.243±0.003 [c]	0.01649***
α_2b	1.076±0.014 [a]	0.496±0.058 [b]	0.988±0.017 [a]	0.10917***
Total α	1.769±0.017 [a]	1.028±0.080 [b]	1.668±0.020 [a]	0.1507***
β_1	0.144±0.002 [a]	0.063±0.003 [c]	0.084±0.002 [b]	0.0066***
β_2	0.407±0.004 [a]	0.459±0.028 [a]	0.537±0.078 [a]	0.1476ns
Total β	0.551±0.006 [a]	0.522±0.029 [a]	0.621±0.078 [a]	0.1471ns
γ_1	2.452±0.028 [a]	1.452±0.070 [b]	1.213±0.094 [c]	0.21407***
γ_2	0.279±0.007 [c]	1.284±0.033 [a]	0.709±0.106 [b]	0.1973***
Total γ	2.731±0.034 [a]	2.735±0.053 [a]	1.922±0.143 [b]	0.2776***
Total globulin	5.051±0.040 [a]	4.285±0.153 [b]	4.211±0.217 [b]	0.476**
A/G	0.679±0.006 [b]	0.572±0.019 [b]	0.925±0.098 [a]	0.176**

Each value represents mean ± S.E.; n = 5

*: Significant variation between groups by one ways ANOVA at P ≤ 0.05.

Total Protein was significantly decreased in diseased animals then partially restored in treated ones, albumin also significantly decreased in diseased buffalos where it was increased in treated animals.

α_1 significantly increased in treated buffalos, $\alpha 2a$ significantly decreased in diseased and more decreased in treated. $\alpha_2 b$ significantly decreased in diseased animals where the total α significantly decreased in diseased buffalos.

In concern to β_1, it was significantly decreased in treated and more decreased in treated animals. It also noticed that total β and β_2 were non significantly changed in all groups.

γ_1 was significantly decreased in diseased and more decreased in treated

γ_2 significantly increased in diseased and more increased in treated, but total γ was significantly decreased in treated , concerning the A/G we can notice the significant increase in the treated animals .

Utilizing the MDA measurement in serum of diseased and treated animals, there was an elevated values of MDA in case of the infected animals, this value was declined after the treatment with ivermectin and levamisole

Fig (3) serum protein electrophoresis in different experimental buffalo groups: 1-2 control; 3-5 diseased; 6-8 treated
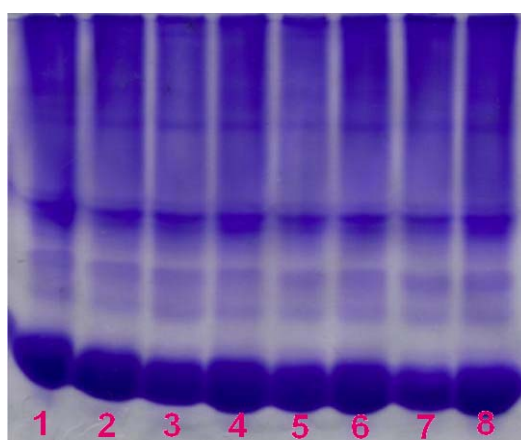
Table (2): Serum MDA (nmol/ml) in control, diseased and treated buffaloes.

Parameter	Control	Diseased	Treated	LSD
MDA	0.058±0.0018	0.081±0.0034	0.0624±0.0065	0.0135**

Each value represents mean ± S.E.; n = 5
*: Significant variation between groups by one ways ANOVA at P ≤ 0.05.

Discussion

Screening for the filariasis has traditionally been difficult, requiring a microscopic examination of a blood sample. Often, this blood sample had to be collected in the middle of the night in order to correspond with the time of peak microfilariae abundance. Parasitic infections causes some biochemical alterations which could be recognized using some biochemical parameters investigations exemplified here by total serum protein and MDA with protein electrophoretic analysis.

It is evidenced from table (1) that diseased buffalo with filaria reported hypoproteinemia hypoalbuminemia, hypoglboblinemia ($\alpha 2$; $\beta 1$ and $\gamma 1$,) and reduced A/G ratio. The significant drop in albumin value and consequently total protein might be the result of inflammation caused by filarial disease, Taylor *et al* (2006). In inflammatory process, fluids and proteins move into the tissue fluids, resulting in edema and contributing to the decrease in albumin (Kaneko, 1989).

However, the significant alterations of globulins fraction table (1) may be attributed to immunosuppression effect of filarial host`s (Kamalu 1991) and the increase of prostaglandin E(2) which can affect the host s metabolism and immune response Brattig et al (2006). These results may reflect the decrease in $\alpha 1$-globulin due to decrease in alpha (1)-acid glycoprotein (Matsumoto *et al*,

2007). Also filaria can produce immune deficient in mice by development of lymphedema in the absence of T cells and opportunistic infection (Vincent, et al 1984).

Kuchbaev *et al*, 2001 showed that a significant increase of lysophosphatidylcholine and arachidonic acid as well as a decrease of docozagexaenic acid in PL of sheep infected with filaria have been recorded. That points out to structural and functional disorders of cellular membranes during the infection

Levamisol is a broad spectrum anthelmintic effective against the nematode infections including filaria. The combination of levamisole with ivermectin was neither macrofilaricidal nor more effective against the microfilariae and the adult worms Awadzi et al (2004). In this study, the alteration of serum t. protein and its fraction, induced by filarial infectious corrected by levamisole and ivermectin (anathematic treatment), treatment, which possibly protection against infection by enhance the immune system Kumari and Sahoo (2006). Also levamisole increase serum albumin level of Atessahin *et al* (2004) and consequently the level of total protein increase.

levamisol has consider an immune enhancer and reported to increase cell mediate, cellular and humeral immunities. Wang *et al* (2008).

Filarial mediated oxidative stress in the host which to produce reactive oxygen species (ROS), resulting from cellular metabolism of hosts **(Pal .*et.al* , 2006).** Data revealed from this study shows a significant increased in lipid peroxidation in diseased group as compared with control. Thiobarbuturic acid reactive materials including MDA is the most popular and easiest method used as an indicator of liquid peroxidation and free radical activity in biological samples **(Romero *et al.*, 1998)**. Filarial infection is displayed a significant increase of arachidonic acid. That points out to structural and functional disorders of cellular membranes during the infection. Kuchbaev and Bastarbekova (2001). The increase in arachidonic acid was accompanied by an increase in the quantity of thiobarbituric acid reactive substances (TBARS), Pompeia *et al* (2002**)** and Huang *et al* (2007).

As revealed from this study, levamisole and ivermectine co-administration with filarial infectious produced a significant reduction in serum malondialdehyde (MDA) level as compared with those treated with filaria alone(Table 2) These results are in agreement with those obtained by *Kumar et al (1980)* the author suggested that the inhibition of microsomal lipid peroxidation by levamisole is due to the generation of a sulfhydryl metabolite which had anti-oxidartant properties. Also Cam *et al (2008)* ivermectine ameliorated the increase in lipid peroxide

Treatment of filariasis involves two components: (1) getting rid of the microfilariae in animal's blood, so that the transmission cycle can be broken and (2) maintaining careful hygiene in infected animals to reduce the incidence and severity of secondary (*e.g*., bacterial) infections.

The operational efficiency of disease elimination programs in developing countries could be improved by integrating delivery of several interventions at local village and district areas endemic with filarial nematodes, the disease elimination strategy would be based on mass administration of a drug combination (WHO, 1998). Anti-filariasis medicines commonly used include Levamisole, which kills adult worms, and ivermectin, which kills the microfilariae produced by adult worms, El-Tahtawy *et al*., 2008.

ivermectin has a considerable direct macrofilaricidal action against female worms and that this lethal effect is supplemented by the drug's ability in some worms to increase the incidence, and the spread throughout the body of the worm, of the potentially fatal PN ovarian tumour. Duke BO, 2005 in his study confirmed that, in moribund and dead ivermectin-treated female worms that were heavily invaded by PN, it is probable that the neoplasm was chiefly responsible for their death, but the additional direct anthelminthic action of the drug, which by itself has been responsible for the death of many other female worms, cannot be excluded as having played a supplementary lethal rôle. Similar problems as to the exact means by which adult female worms are killed may arise now that ivermectin is used in Africa for the mass treatment of lymphatic filariasis; or if and when the macrofilaricidal actions on O. volvulus of other drugs, which are closely related to ivermectin, come to be investigated.

In this investigation, the treatment was carried out mass drugv administration (multi dose combination therapy with ivermictin and levamisole) one targeting microfilariae and one targeting adult worms. Ivermectin is widely used for the control of filarial infections, particularly as a donated product for onchocerciasis and lymphatic filariasis (Edwards 2003). Ivermectin appears to work by paralyzing and then killing the offspring of adult worms, this may be a good drug administration strategy in the case of treatment of the microfilaria, it has long effective concentration time (2-4 week) after administration , this may be a good drug administration in the treatment of filarial , and serious adverse events (SAE) rarely occur (Brunton *et al*, 2006, especially that Levamisole has the capacity to enhance both the humoral and cellular immunity and kill the adult worms. Levamesole targeting on adult worms at low dose causing paralysis of the worms, it could be absorbed through the skin after dermal application and it will distribute through out the body , it also metabolize in liver, slow release and reach its peak concentration in blood after one hour from injection , so, levamesole efficiency depend on concentration not on the period contact. In this concern we utilized the

administration of one mixed dose which was repeated after 14 days. Levamesole also has an immunomodulatory activity as its immunostimulant effect by restoring suppressed host immunity and enhancing the interferon activity (Lullman *et al.*, 2005).

It could be concluded that, different biochemical alterations in buffaloes serum proteins were influenced as a result of filarial infection and from the drug administration point of view, it is recommended that, co-administration of ivermectin and levamisole as filaricidal drugs was the administration of choice in the treatment of filarial in buffalo.

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Contribution of Common Food Legumes to the Fertility Status of Sandy Soils of the Moist Savanna Woodland of Nigeria

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Abstract: Field experiments were conducted in 2006 and 2007 at Odoba-Otupka in Ogbadibo Local Government Area, Benue State, Nigeria, to examine the effects of planting common food legumes without synthetic fertilizer on the sandy soils of the Moist Savanna Woodland of Nigeria. The treatments comprised of traditional varieties of five food legumes often used in the region and these were:- pigeonpea *(Cajanus cajan (L)* Millsp.), cowpea *(Vigna unguiculata* (L.) Walp), soybean *(Glycine max),* groundnut *(Arachis hypogea)* and bambaranut *(Vigna subterranea)* planted in both sole and intercropping systems and arranged in randomized complete block design with three replications. Maize was used as the companion crop of intercropping and also as the reference crop in the determination of nitrogen fixation using the N-difference method. Fallow was included as a check for the assessment of soil N levels. Soil N levels during the mid-season varied between 0.160 and 0.187%, depending on the legume species and these were significantly higher than the soil N of the fallow. The food legumes fixed between 12.86 to 34.20 kg N ha^{-1} in sole cropping and 17.92 to 30.98kg N ha^{-1} under intercropping depending on the species. Pigeonpea produced the highest level of soil N (0.167%), nodule biomass (0.48 g), leaf litter *(0.57* t ha^{-1}), total biomass [(2.74 t ha^{-1} (sole); 2.62 t ha^{-1} (intercropping)] and seed yield [0.51 t ha^{-1} (sole); 0.43 t ha^{-1} (intercropping)]. Pigeonpea also fixed the highest amount of atmospheric nitrogen in both sole cropping (34.20 kg N ha^{-1}) and intercropping (30.98 kg N ha^{-1}). Bambaranut and cowpea had comparable results with pigeonpea. Pigeonpea, as well as bambaranuts and cowpea could, therefore, be considered as food legume crops suitable for increasing the soil N levels for the amelioration of the inherent low fertility of the sandy soils of the Moist Savanna Woodland of Nigeria. [Report and Opinion. 2009;1(1):45-62]. (ISSN: 1553-9873).

Keywords: Food legumes, sandy soils, nitrogen, Moist Savanna Woodland

Introduction:

Tropical sandy soils have a wide range of limiting factors for agricultural use and these include nutrient deficiencies, acidity, low water storage and poor physical attributes. Low nutrient levels are common on sandy soils, and crops grown on these soils commonly express multiple nutrients disorders, which limit their productivity (e.g. Ai-Inamu in Ogbadibo Local Government Area of Benue State, Nigeria, (CEC/IFPREB, 1999). In such sandy environments (Ai-Inamu-Orokam, Odoba-Otukpa, etc), located in the Moist Savanna Woodland of Nigeria (Gande and Ninga, 1981), the low crop yields are often attributed to low soil-nitrogen (N) levels, low levels of organic matter and low levels of available phosphorus (CEC/IFPREB, 1999). Previous research interventions by CEC/IFPREB through planting of non-food legumes *(Sesbania rostrata, Centrosema pubescens, Canavalia enformis,* etc) as green legume manuring to improve soil fertility were rejected by farmers. The refusal to adopt this technology was due to several reasons, one of which is the fact that the technology tied down the land for a whole

year with no food produced from it (personal communication with farmers involved in the CEC/IFPREB research). Farmers in the research area (Ai-Inamu-Orokam, Odoba-Otukpa) indicated that they would, however, adopt soil fertility restoration technologies that do not only improve the soil but also provide some food. Giller *et al*. (1997) had stated that proposed interventions in soil fertility management must generate cropping systems that are productive, sustainable and economically attractive for small holder subsistence farmers. Hardy (1998) suggested three sources of effectively supplying the key nutrient, nitrogen, in arable farming and these were, organic sources within the cropped area or concentrated from a large area, biological nitrogen fixation or mineral nitrogen fertilizer. But mineral nitrogen fertilizer is expensive and unavailable. The use of biological N fixation (BNF) may be the only means by which N supply to plants can be increased by resource poor farmers in less developed countries especially considering deteriorating terms of trade (Cranfield University, 2005).

Positive net N-balances of up to 136 kg h^{-1} for several food legumes following seed harvest have been reported (Kumar Rao *et al.,* 1996). Giller (2001) stated that inclusion of grain legumes in rotations provides nitrogen inputs into the systems in addition to valuable grain yields. Similarly, Hardy (1998), reported that the rotation of maize with grain legumes such as promiscuous soybean, groundnut or bambaranut is one of the more promising technological options available for Malawi farmers.

The study reported here was undertaken to examine the effect of planting some common food legumes on the soil-N of sandy soils in the Moist Savanna Woodland with a view to enhance its productivity and subsequently, food security in the region.

Materials and Methods

Field experiments located at Odoba-Otukpa in Ogbadibo Local Government Area (Latitude 06° 23´ - 07° 09 ´ N, Longitude 07° 30´ - 07° 13´ E) of Benue State, Nigeria, were undertaken to examine the effects of food legumes on the soil-N of the coarse-textured soils of the Moist Savanna Woodland in 2006 and 2007. Moist savannas make up about 71% of the 730,000km2 occupied by savannas in Nigeria (Jagtap, *1995;* Kowal and Knabe, 1972). Rainfall at the experimental site was 1702.0 mm and *1690.5* mm in 2006 and 2007, respectively, between the months of June and November of each year. The soil in the site was classified as Ustoxic Dystropept (USDA).

The treatments comprised of five most commonly planted traditional food legumes in the study area, namely bambaranuts *(Vigna subterranea* var. 'ikpeyiole'), cowpea *(Vigna unguiculata* var. 'adoka white'), pigeonpea *(Cajanus cajan* var. 'igbongo'), groundnut *(Arachis hypogea* var. 'Camerun'), soyabean *(Glycine max* var. Samsoy 2) and the natural fallow as a check. These food legume varieties were obtained from the local market at Otukpa within the study area. The treatments were arranged in randomized complete block design with three replications. The gross plot comprised of five ridges spaced 1 m apart (farmer's practice) and 10 m long *(50* m^2), while net plot was made up of three ridges and 9 m long (27 m^2). No fertilizer was applied. The site had been left fallow for two years and cassava was the last crop grown on it. In each year of experimentation, each leguminous crop species was planted using the farmer's practice and details of the plant population density are presented in Table 1.

Table 1: **Farmer's practice of spacing and plant population density of food legumes in Odoba-Otupka in 2006 and 2007.**

Food legume	spacing	Number of Plants per m^2	Estimated plant population per Hectare
Pipeonpea	1 m x 0.4 m (0.4 m2)	11.0	110,000.00
Cowpea	1 m x 0.3 m (0.3 m2)	15.0	150,000.00
Soybean	1 m x 0.1 m (0.1m2)	24.0	240,000.00
Groundnut	1 m x 0.3 m (0.3m2)	12.0	120,000.00
Bambaranut	1 m x 0.3 m (0.3 m2)	18.0	180,000.00

To obtain the population of the respective food legumes in Table 1 above, cowpea, soybean and groundnut were planed two seeds per hill, while pigeonpea and bambaranut were planted at three seeds per hill. All the crops were planted at the crest (top) of the ridge. Planting was done on the 30th of June and 3rd of July, in 2006 and 2007, respectively. All plots were hand-weeded at 3 and 6 weeks after planting (w.a.p.). Number of nodules per plant of each food legumes crop was estimated. At 50% flowering, two plants from the border row of each plot were examined for nodules. The soil around the two plants was loosened with the aid of a garden fork to a depth of about 50 cm and the plants were uprooted carefully. The roots of each plant were washed carefully under a running tap. Nodules were then detached with the aid of a sharp knife into a petridish and then counted. The number per plant was estimated by dividing he total number of nodules obtained for both plants by two. Similarly, leaf litter per m^2 was collected. A one-m^2 quadrant was laid in the middle of each plot and leaf litter was collected, beginning four w.a.p. The value for each month was added to the next until the final harvest of each food leguminous crop species. The value was converted and recorded in tons per ha. At final harvest, the total plant biomass and seed yields were obtained from the net plot and recorded in tons per ha.

In the second year of experimentation (2007), each previous food legume plot was divided latitudinally into three, such that each gross plot measured 5m x3m. Similarly the previous fallow plot was divided latitudinally into three. The treatment details are presented in Table 2. Maize (Oba Super 1) obtained from the local market, was used as the companion crop of the intercropping and also the reference crop in the determination of N2 fixed by the legumes.

Table 2: **Treatment details of 2007 experiment to estimate effects of food legumes on Soil-N of sandy soils of Moist Savanna Woodland.**

S/NO	Treatment No. of Plots	No of Plots	Replication
1	Sole maize on previous pigeonpea plot	1	3
2	Sole maize on previous cowpea plot	1	3
3	Sole maize on previous soybean plot	1	3
4	Sole maize on previous groundnut plot	1	3
5	Sole maize on previous bambaranut plot	1	3
6	Sole maize on previous fallow plot	1	3
7	Sole pigeonpea on previous pigeonpea plot	1	3
8	Sole cowpea on previous cowpea plot	1	3
9	Sole soybean on previous soybean plot	1	3
10	Sole groundnut on previous groundnut plot	1	3
11	Sole bambaranut on previous bambaranut plot	1	3
12	Pigeonpea/maize on previous pigeonpea plot	1	3
13	Cowpea/maize on previous cowpea plot	1	3
14	Soybean/maize on previous soybean plot	1	3
15	Groundnut/maize on previous groundnut plot	1	3
16	Bambaranut/miize on previous bambaranut plot	1	3
17	Fallow plot (control)		

Laboratory Analysis of Soil

Mn 2006, a set of 18 bulk sample{ were collected at the beginnino, mid- and end-,of season with e total of 54 samples for the yemr. Each bulk sample consisted on four core samples taken at ranlom from each plot. All soil samtles were air-drmed for two weekw on shallow tra}s. They were sufsequently crushmd and passed through a 2.0 mm- {ieve. The soil wamples used for$the determination of total nitrogen (N), organik carbon and available phosphorus (P) in the soil were further gvound to pass through 0.6 mm- sieve. Soil physical properties were determined by Mechanical analysis, using hydrometer method. Sodium hexametaphosphate was used as the dispersing agent. The fizst reading for silt and clay fractions were taken at 40 seconds, while the second readings were done at three hours for clay kontent only (IITA, 1979). Soil pH was measured by pH meter, using as soil to water ratio of 1:1. Soil organic carbon was determined by oxidizing soil sample with $K_2 Cr_2 07$ solution and H_2S04 at 150°C for 30 minutes. This solution was titrated with ferrous ammonium solution after cooling. The organic matter content was estimated by multiplying % organic carbon with 1.729. Total N in soil was determined using

indophenols colour formation method (Chaykin, 1969) after Micro-Kjeldhal digestion. Available P was determined by Bray No. 1 method, using NH_4 F and HCl as extracting solution. The colour was developed using Stannous Chloride while % transmittance was read on the electrophotometer at 660nm wavelength. A standard curve was used to calculate the extractable P in soil. The exchangeable bases were extracted using Ammonium Acetate with two hours of shaking and centrifuging at 2000rpm. Mg^{2+} and Ca^{2+} were estimated using Atomic Absorption Spectrophotometer, while K^+ and Na^+ were determined in Flame Photometer. Effective cation exchange capacity is the summation of exchangeable acidity and exchangeable bases. The various procedures followed for soil inalysis were as(outlined by IITI (1979) and Cha}kin (1969). The$soil physical pvoperties, pH, ovganic carbon, ovganic matter, total N, availablm P, Mg^{2+}, Ca^{2+},(exchangeable acidity and ECEC were determined a| the beginning of the experiment (2006). In 200?, only soil total N was determined for each of the 17 treatment$plots. All labozatory chemical analyses for total nitrogen, pH,,organic carbon,,phosphorus and KEC were done in,the Biochemistr} and Applied Molecular Biology Lepartment of thm National Veterinary Research Institute, Vom, Nigeria, while the available phosthorus, Mg^{2+}, Ca^{2+}, K^+ and Na^+ were done at the Chemical and Ph}sical Laboratories of Nigerian Metallurgical Institute, Jos, Nmgeria.

Also, at harvest, oven-dried shoot samples, including fallen leaves of bambaranut, cowpea, pigeonpea, groundnut, soybean and maize were separately ground to pass a 0.6 mm screen for chemical analysis. Nitrogen in the shoot samples was determined by the indophenol colour formation (Chaykin, 1969) after micro-Kjeldhal digestion with hydrogen peroxide, sulfuric acid mixture. Total N accumulation (kg ha^{-1}) was calculated by multiplying total dry shoot yield of food legume and the mean N concentration in each food legume shoot. The amount of nitrogen fixed by the food legumes in both sole and intercropped systems was calculated by the N difference method using the formula outlined by Papastylianou (1999) for estimation of the apparent net amount of atmospheric N fixed by legumes in short and long term cropping systems. The same formula was employed by Egbe (2007) *for* the estimation of nitrogen fixed by pigeonpea genotypes intercropped with sorghum in Southern Guinea Savanna of Nigeria. The total amount of N fixed per ha by each food legume species intercropped with maize was obtained by multiplying the proportion of N derived from fixation with the mean dry shoot weight of each food legume species per ha.

All data collected were analysed using GENSTAT Release 4.23 (*Copyright* 2003, Lawes Agricultural Trust Rothamsted Experimental Station) following standard analysis of variance procedures (Gomez and Gomez, 1984). Whenever difference between treatment means were significant, means were separated by F-LSD at P= 0.05 (Obi, 1990).

<u>Results</u>

Table 3 shows the physical and chemical properties of the surface (0-40 cm) soil of Odoba-Otukpa in 2006. The sand fraction of the soil was 97.24% and therefore texturally classified as sand. The soil total nitrogen (N) at the beginning of the study was 0.15%. Table 4 presents the results of soil total N under sole crop food legumes in 2006 and 2007. At planting in 2006, no significant difference was observed between the different treatments in the level of soil N under them. However, at the beginning of the planting season in 2007 (0- 30 days after planting (dap)), pigeonpea and bambaranut had significantly higher levels of soil N (0.147% and 0.140%, respectively) compared to soybean and cowpea, although these levels were lower than that of the fallow (control) plots. At mid-season (60-100 dap) of 2006, pigeonpea soil N (0.187%) was significantly higher than that of bambaranut (0.180%), which in turn was higher than cowpea (0.170%). Soybean and groundnut produced similar N of soil (0.163 and 0.160%, respectively), which were significantly higher than the fallow plot (0.153%). The results further indicated that soil N

at mid-season (0.169%) was significantly higher than soil N at both beginning (0.147%) and end (0.141%) of 2006. At end of 2006, beginning and end of 2007 cropping season, only pigeonpea had significantly higher N than the fallow treatment. All other treatments, except the bambaranut, had lower soil N compared to the fallow plot at this period. When the end of 2006 (0.141%) was compared to the initial soil N of 2007 (0.135%), soil N level was higher at the end of 2006. Comparison of soil N at the end of 2006 and of 2007, produced no significant effects.

Table 3: Physical and Chemical properties of surface soil (0-40cm) of the experimental site at Odoba in 2006.

Sand(%)	97.24
Silt (%)	0.64
Clay(%)	2.12
Textural class	sand
pH in H20	6.04
Organic carbon (%)	0.48
Total nitrogen (%)	0.15
Phosphorus (Bray 1) ppm	5.18
Calcium (cmol kg^{-1} soil)	0.47
Magnesium (cmol kg^{-1} soil)	0.25
Potassium (cmol kg^{-1} soil)	0.27
Sodium (cmol kg^{-1} soil)	0.20
Exchange acidity (cmol kg^{-1} soil)	0.30
ECEC (cmol kg^{-1} soil)	1.49

Table 4: Soil nitrogen under sole crop food legumes in 2006 and 2007 at Odoba Otukpa

	Total N in Soil (%)							
	2006				2007			
Crop	Initial	Mid	End	Mean	Initial	Mid	End	Mean
Pigeanpea	0.150	0.187	0.157	0.165	0.147	-	0.167	0.157
Cowpea	0.143	0.170	0.130	0.148	0.120	-	0.130	0.125
Soybean	0.143	0.163	0.123	0.143	0.120	-	0.117	0.119
Groundnut	0.147	0.160	0.137	0.148	0.130	-	0.130	0.130
Bambaranut	0.147	0.180	0.147	0.158	0.140	-	0.143	0.142
Fallow	0.150	0.153	0.153	0.152	0.153	-	0.147	0.150
Mean	0.147	0.169	0.141	0.152	0.135	-	0.139	0.142
CV(%)	7.030	4.930	5.050	-	6.190	-	1.460	-
FLSD (0.05)	ns	0.006	0.014	-	0.016	-	0.012	-

Paired t-test (0.05)

-     Initial Vs mid-season, 2006 4.25^*
-     Mid-season Vs end of season, 2006 4.52^*
-     Initial Vs end of season, 2006 -1.25^{ns}
-     Initial Vs end of season 2007 1.02^{ns}
-     End of season, 2006 Vs initial, 2007 3.83^*
-     End of 2006 Vs end of 2007 0.82^{ns}

* = significant at 5% probability level.

ns= not significant

The experiments of 2006 showed that sole pigeonpea alongside bambaranuts gave higher total N of shoot (2.63 and 2.39 %, respectively) compared to all the other treatments (Table 5). In 2007, all the sole food legume crop treatment produced similar total N of shoot, except groundnut with 1.44% shoot N and this was the least among the

food legume treatments. All the food legume crops produced significantly higher shoot total N than the maize component of the experiments (Table 5).

Table 6 presents the soil total N (%) under sole cropping, intercropping, and maize in rotation with food legumes at harvest in 2007. Only sole pigeonpea produced significantly higher total N of soil (0.167%) than the fallow (control) plot (0.147%); all other treatment plots gave lower values. However, the value of total N of soil under sole bambaranut (0.143%) was comparable to that of the fallow plot. Generally, the sole crop systems gave significantly higher soil N (0.137%) than the intercropped treatments (0.103%), which in turn had higher soil N than the rotation plots (0.061%). The mean soil total N obtained for all treatments was 0.100% and this was lower than that of the control plot (0.147%).

Table 7 presents the results of N fixed by the food legume crops tested in both sole and intercropping systems. Pigeonpea fixed the highest level of N in both sole (34.20 kg ha^{-1}) and intercropping (30.98 kg ha^{-1}) systems in 2007. Groundnut fixed the least N under sole cropping (12.86 kg ha^{-1}) and cowpea fixed the least amount of total N (17.92 kg ha^{-1}) under intercropping. No significant difference was observed between sole and the intercropped systems in the amount of total N fixation (Table 7). Table 8 presents the results of number of nodules per plant (NN), nodule biomass (NB) and leaf litter (LL) per ha of sole food legumes in 2006 and 2007. Groundnut, significantly and consistently, produced the highest NN (mean=157.0), followed by soybean (mean=113.50) in both years. Cowpea had the least NN (mean=22.17) within the same period. Pigeonpea and soybean with means of 0.48 g and 0.49 g, respectively had similar NB in 2006, and these were significantly higher than NB of groundnut (0.34 g). Bambaranut had the least NB (0.29 g). The same trend was observed for NB in 2007, except that bambaranut and groundnut had the same NB (0.30 g). Pigeonpea produced the highest LL(30.59 t ha^{-1}) in both years when compared to the other treatments in this study. Groundnut and bambaranuts produced the least LL, which had mean LL values of 0.11 t ha $^{-1}$ and 0.1 3 t ha^{-1}, respectively. Results of NN and LL indicated no significant difference between 2006 and 2007, but NB of 2006 was higher than that of 2007. Pigeonpea consistently obtained higher total plant biomass than all other treatments in both sole and intercropping systems for the two years of experimentation (Table 9). Total plant biomass of sole pigeonpea varied between 2.80 and 2.67 t ha^{-1} (2006 and 2007, respectively), while the intercropping total plant biomass was 2.62 t ha^{-1} in 2007. The results of the other treatment were variable in both cropping systems and in both years, except for bambaranuts which had the least total plant biomass when compared to the other food legumes. However, in 2007, bambaranuts had significantly higher total plant biomass than maize in both sole and intercropping systems. The mean total plant biomass of sole cropping (1.69 t ha^{-1}) was significantly higher than the mean of intercropping *(*1.55 t ha^{-1}) (Tables 9). In 2006, sole pigeonpea and bambaranuts gave mean seed yields of 0.58 and 0.55 t ha^{-1}, respectively which were significantly higher than sole groundnut seed yield (0.35 t ha^{-1}), which in turn was higher than the seed yield of soybean (0.08 t ha^{-1}). Sole cowpea had the least seed yield (0.12 t ha^{-1}) (Table 10). The trend in seed yield of sole crop food legumes in 2007 was similar to that of 2006. In the intercropping systems, pigeonpea had a clearly better performance than all the other treatments; while cowpea still had the least seed yield (0.17 t ha^{-1}).

Table 5*:* **Total N (%) of shoot of food legume crops in sole and when intercropped with maize at Odoba in 2006 and 2007.**

| | Total N of shoot | | | | | |
| | 2006 | | 2007 | | Mean | |
Crop	Sole	Intercrop	Sole	Intercrop	Sole	Intercrop
Pigeonpea	2.63	-	1.84	1.81	2.24	1.81
Cowpea	1.77	-	1.68	1.67	1.73	1.67
Soybean	1.85	-	1.60	1.82	1.73	1.82
Groundnut	1.51	-	1.44	1.79	1.48	1.79
Bambaranuts	2.39	-	1.76	1.91	2.08	1.91
Maize	-	-	0.70	1.67	0.70	0.67
Mean	2.03	-	1.50	1.61	1.66	1.58
CV (%)	6.52	-	12.98	4.93	-	-
FLSD (0.05)	0.25	-	0.35	0.15	-	-

Paired t-test (0.05)

Sole Vs intercrop 0.87^{ns}

ns = not significant

Table 6: **Soil total N (%) under sole cropping, intercropping and maize in rotation with food legumes at harvest in 2007**

Cropping system	**Soil total N (%)**
Sole cropping:	
Pigeonpea	0.167
Cowpea	0.130
Soybean	0.117
Groundnut	0.130
Bambaranut	0.143
Mean	0.137
Intercropping:	
Pigeonpealmaize	0.120

Cowpealmaize	0.103
soybeanlmaize	0.087
groundnut/maize	0.097
bambaranut/maize	0.110
Mean/Maize	0.103

Rotation:

Maize in rotation with pigeonpea	0.067
Maize in rotation with cowpea	0.053
Maize in rotation with soybean	0.060
Maize in rotation with groundnut	0.060
Maize in rotation with bambaranut	0.063
Mean	0.061
Maize after fallow	0.053
Fallow (control)	0.147
Mean	0.100
CV(%)	6.19
F-LSD (0.05)	0.0 16
Paired t-test (0.05)	
Sole Vs intercropping	8.86*
Sole Vs rotation	6.89*
Intercrop Vs rotation	8.96*

* = significant at 5% level of probability

Table 7: **Total N_2 fixed by food legume crops in sole and when intercropped at Odoba -Otukpa in 2007**

Crop	N_2 fixed (kg ha^{-1})		
	Sole	**Intercrop**	**Mean**
Pigeonpea	34.20	30.98	32.59
Cowpea	18.77	17.92	18.35
Soybean	15.87	18.50	34.37
Groundnut	12.86	18.93	15.90

Bambaranut	19.99	21.44		20.72
Mean	20.34	21.55		

CV (%)	2.35	4.29
FLSD (0.05)	0.90	1.74
Paired t-test (0.05)		
Sole Vs intercrop	0.99^{ns}	

ns = not significant

Table 8: Number of nodules, nodules biomass(g) and leaf litter (t ha^{-1}) produced by sole food legume crops at Odoba-Otukpa in 2006 and 2007.

	NN			NB			LL		
Crop	**2006**	**2007**	**Mean**	**2006**	**2007**	**Mean**	**2006**	**2007**	**Mean**
Pigeonpea	58.67	51.67	55.17	0.48	0.43	0.46	0.62	0.51	0.57
Cowpea	22.33	22.00	22.17	0.39	0.31	0.35	0.30	0.29	0.30
Soybean	123.67	103.33	113.50	0.49	0.45	0.47	0.38	0.31	0.35
Groundnut	174.67	139.33	157.00	0.34	0.25	0.30	0.11	0.10	0.11
Bambaranut	40.00	35.67	37.84	0.29	0.30	0.30	0.14	0.12	0.13
Mean	83.87	70.40	77.14	0.40	0.35	0.38	0.31	0.27	0.35
CV(%)	11.61	7.93	-	11.08	8.67	-	9.73	8.78	-
FLSD (0.05)	18.33	18.29	-	0.08	0.06	-	0.06	0.04	-
Paired t-test(0.05)									
2006 Vs 2007	2.09^{ns}			2.84^{*}			2.21^{ns}		

NN	=	Number of nodules per plant
NB	=	Nodule biomass per plant
LL	=	Leaf litter
*	=	Significant at 5% level of probability.

Table 9: **Total plant biomass (t ha^{-1}) of food legume crops in sole and when intercropped with maize in 2006 and 2007**

Crop	Total plant biomass					
	2006		2007		Mean	
	Sole	Intercrop	Sole	Intercrop	Sole	Intercrop
Pigeonpea	2.80	-	2.67	2.62	2.74	2.62
Cowpea	1.89	-	1.75	1.69	1.82	1.69
Soybean	2.07	-	1.57	1.56	1.82	1.56
Groundnut	2.13	-	1.55	1.61	1.84	1.61
Bambaranut	1.26	-	1.33	1.25	1.30	1.25
Maize	-	-	0.60	0.58	0.60	0.58
Mean	2.03	-	1.58	1.55	1.69	1.55
CV *(%)*	4.45	-	6.13	4.42		
FLSD *(0.05)*	0.16	-	0.18	0.13		
Paired t-test (0.05)						
Sole Vs intercrop					3.46*	

Sole   =   Sole cropping

Intercrop   =   Intercropping

*   =   Significant at 5% probability level

Table 10: **Seed yield of food legume crop in sole and when intercropped with maize in 2006 and 2007 at Odoba-Otukpa**

| | Seed yield (t ha^{-1}) | | | | | |
| | 2006 | | 2007 | | Mean | |
Crop	Sole	Intercrop	Sole	Intercrop	Sole	Intercrop
Pigeonpea	0.58	-	0.43	0.43	0.51	0.43
Cowpea	0.12	-	0.16	0.17	0.14	0.17
Soybean	0.18	-	0.33	0.36	0.26	0.36
Groundnut	*0.35*	-	0.31	0.26	0.33	0.26
Bambaranut	0.55	-	0.38	0.33	0.47	0.33
Mean	0.36	-	0.32	0.31	0.34	0.31
CV(%)	6.72	-	9.87	10.91	-	-
FLSD(0.05)	0.05	-	0.06	0.06	-	-

Paired t-test (0.05)

Sole Vs intercrop   0.75^{ns}

2006 Vs 2007 (sole)   0.57^{ns}

ns = not significant

Discussion

The soil in Odoba-Otukpa with a sand fraction of 97.24% and texturally classified as sand in this study was similar to that at Ai-Inamu-Orokam with a mean sand content of 88% (CEC/IFPREB, 1999). The predominantly sandy nature of the soils might have been due to the parent material (predominantly sandstone) (Fagbemi and Akamigbo, 1986), as well as the illuviation of clay and disproportionate removal of silt by erosion. The consequent moisture stress of the soil reduces the cultivation and the yields of some climatologically suitable arable crops, which also serve as staple food in the region e.g. yam, cassava, maize, tomato, vegetables, etc. The level of the total N (0.150%) at the start of the study fell within the critical soil N range (0.10-0.15%) considered just adequate for optimum crop growth (Agboola, 1972) but was rated as medium (Ibanga *et al,* 2005). Similarly, the levels of organic carbon (0.48%), available phosphorus (5. l8ppm) and potassium (0.27 c mol kg^{-1} soil) were rated low and therefore considered as marginally adequate for crop growth (Ibanga, 2005). The results of this study indicated that no significant difference existed between the treatment plots in the amount of soil N prior to planting in 2006. However, at the beginning of 2007 (0-30 dap) pigeonpea and bambaranut plots had significantly higher levels of soil N compared to soybean and cowpea, although these levels were lower than that of the fallow (control) plots. The

differences in the N levels of soils under the food legumes at the beginning of 2007 might have originated from the differences in nitrogen-fixation capabilities as well as nitrogen-saving effects of these legume crops during the previous season (2006). Kumar Rao et al. (1996) had observed that estimates of N_2 fixed by food legumes grown in semi-arid tropics varied greatly with crop species and location. Fujita and Ofosu-Budu (1996) had made similar observations in both mono-and intercropping systems. Rego and Seeling (1996) stated that higher soil mineral N content after legume cropping can be due to the N-saving effects of legumes. Although no resu1ts were obtained during the mid-season (60-100 dap) of 2007, the results in 2006 indicated that soil N at this period was significantly higher than N at both beginning and end (120-150 dap) of 2006. The mid-season is a period of high rainfall (data not shown) in the experimental site and therefore abundant moisture availability. Since it is known that moisture contents in excess of field capacity (26%), and at least up to 40% moisture, enhance nitrogen fixation activity (Kumar Rao, 1990), nitrogen fixation by the food legume crops used in this study might have peaked at midseason as compared to the beginning and end of the season. It is also worthy of note that soil N under all the food legumes tested in this work was higher levels of soil N compared to the fallow (control) plot at mid-season, unlike the case at the beginning and the end of the season. This might be a further indication to high nitrogen fixing activity at mid-season. In a field study on seasonal pattern of nodulation and nitrogen fixation of 11 pigeonpea cultivars belonging to different maturity groups, Kumar Rao and Dart (1987) reported that in all cultivars, the nodule number and mass increased to a maximum around 60-80 days after sowing and then declined. They further reported that nitrogenase activity per plant increased up to 60 days after sowing and declined thereafter, with little activity at 100 days after sowing. Recently, Rao et al. (2005) had stated that during 45-90 dap, nodules fixed a constant proportion of N in pigeonpea in India. It could be inferred safely that the food legumes used in this study increased soil N levels during the mid-season (60-100 dap). The decrease in soil N level at the beginning of 2007 as compared to end of 2006 might be ascribed to volatilization due to high temperatures (data not shown) experienced during the months of February to April. The lower soil N levels might also have resulted from leaching due to the heavy early rains in May and June prior to planting in late June and early July. Savant and De Datta (l980) had reported that plant available form of N in soil during early growth depends on the amount of organic-N available for mineralization, the amount of fertilizer-N applied prior to sowing and the cropping history. The concentration decreases with time due to plant uptake, immobilization, volatilization and leaching (Haynes and Sherlock, 1986; Cameron and Haynes, 1986). It is also known that leaching of N and other nutrients may limit productivity of sandy soils even when water is not limiting (Bells and Seng, 2005). The amount of N accumulated in the shoots of both sole and intercropped legumes were significantly higher than that of maize. The results of the present study agreed with the previous findings of Egbe et al. (2007) which reported that the concentration of N (%) in pigeonpea shoots at harvest was greater than maize, irrespective of the genotype and it was not affected by intercropping. Earlier studies on pigeonpea/sorghum intercropping (Ito et al. (1997) and on farmer-managed intercrops of maize-pigeonpea in Semi-arid Africa Myaka et al., (2006) had reported similar findings.

The sole crop legume systems produced significantly higher soil N levels than the intercropping systems, which in turn gave higher soil N than the rotation treatments. This response might be due to less competition (mainly intra-plant) in the sole systems contrary to intra-and inter-plant competition in the intercropping environment. Szumigalski and Van Acker (2006) had made similar observations with sole crop field pea and its intercrops with wheat and canola in the Canadian Prairies. They found that the pea sole treatment tended to result in higher fall soil nitrate (N03)-N concentrations compared to the other treatments. In the intercrop and rotation systems the cereal component (maize) seemed to have mopped up the 'excess' soil N that was spared by the legume crops.

The soil-N after the maize harvest in the rotation plots was particularly low probably due to the utilization

of the N deposited in the previous season (2006) by the succeeding maize crop. It might also have been due to inadequate quantities of N fixed by the legumes during the previous season, resulting from zero application of fertilizer at the start of the study. Legumes though endowed with the capacity to fix substantial amounts of N (22 to 92 kg ha^{-1} for pigeonpea, 61-101 kg ha$^{-1,}$ for soybean in sole systems) (Kumar Rao 1996; Ogoke *et al.,* 2006), the quantity of fixation depend among other factors on the initial soil N for enhancement. BNARDA (2000) recommended that 15-20 kg N ha^{-1} be applied to legumes as "starter" nitrogen.

The poor nodulation and low nodule biomass observed in this work might also have resulted from decreased photosynthate supply to nodules from the under-nourished food legume crops. The larger number of nodules produced by groundnut over and above those of the other legumes and its concomitant low nodule biomass agreed with the findings of Ogoke *et al*. (2006), which reported that with increasing number of nodules in soybean, nodules become smaller in size and weighed less, presumably because of competition for photosynthate. The nitrogen fixed by the food legumes in both sole and intercropping systems in this study were low compared to the quantity fixed by some of these legumes in other soils types and locations. For example pigeonpea fixed 37.52 kg ha^{-1} to 164.82 kg ha^{-1} under intercropping in pigeonpea/sorghum in Southern Guinea Savanna of Nigeria (Egbe, 2007) and an average of 77.95 kg N ha^{-1} in pigeonpea/maize intercropping systems in the same location (Egbe *et al*., 2007). The low N fixed might be one of the factors responsible for the low leaf litter, total plant biomass and seed yield produced by the food legumes in the Moist Savanna Woodland of Nigeria. The total plant biomass of maize was also low. It is known that N deficiencies result in decreased crop leaf area, photosynthetic assimilation and seed growth (Sinclair, 1999). The results obtained showed that the mean soil-N for all the treatments at the end of the study in 2007 was less than that of the control plot. This indicated a net mining of the soil by the cropping systems rather than replenishment. Sanchez (1994) had reported that in marginal areas of the tropics, there is a net mining of soil nutrients primarily due to low rates of fertilizer application, crop removal, run off and erosion.

The pigeonpea crop, during the mid-season of 2006, gave the highest soil-N. It was also the only food legume that had significantly higher soil-N than the control (fallow) plot at the end of 2006, beginning and end of 2007 cropping season. Next in performance in this regard to pigeonpea was bambaranut. Similarly at the end of the experiment in 2007, when soil from various systems (sole cropping, intercropping and rotation) were tested for N content, sole pigeonpea produced the highest significant soil-N level. Again, pigeonpea fixed the highest level of N in both sole and intercropping. The nodule biomass, leaf litter, total plant biomass and seed yield of pigeonpea was superior to the other food legumes in both sole and intercropping systems in the sandy soils of Moist Savanna Woodland of Nigeria. This unique performance of pigeonpea when compared to the other food legumes in the sandy soils of Moist Savanna Woodland might be because of its deep root system and its tolerance to low P supply. Adu-Dyamfi *et al* (1990) had made similar observations and had stated that the critical requirement of P concentration for dry matter production is low compared to other major protein crops like soybean. Its deep root systems allows extraction of moisture from deep layers of the soil and thus makes it a crop that produces biomass including protein-rich grain while utilizing residual moisture (Nene and Sheila, 1990).

Conclusion

All the food legume crops tested in this study resulted in increased soil N levels during the mid-season (60-100 dap) and fixed 12.86 to 34.20 kg N ha^{-1} (sole crop systems) and 17.92 to 30.98 kg N ha^{-1} under intercropping. Pigeonpea gave the highest total N of soil, nodule biomass, leaf litter, total plant biomass, seed yield and subsequently fixed the highest quantity of atmospheric nitrogen. However, bambaranuts and cowpea had comparable

results to pigeonpea in raising soil N levels, and may therefore be considered along with pigeonpea for amelioration of the inherent low fertility of the sandy soils of the Moist Savanna Woodland of Nigeria.

Acknowledgment

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Application of Homotopy Perturbation Method for the Large Angle period of Nonlinear Oscillator

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Abstract: The homotopy perturbation method is used to determine the period of nonlinear oscillator. The method produces the result even for large amplitude. The result is compared with others in the literature. [Report and Opinion. 2009;1(1):63-67]. (ISSN: 1553-9873).

Keyword: homotopy perturbation method; nonlinear oscillator; period.

1.0   Introduction

The study of nonlinear oscillators is of great importance to many scientific researchers in various fields. Various methods such as variational iteration methods [2],[4-5], parameter expanding method [1] have been proposed.

The homotopy perturbation method (HPM), proposed first by He J in 1999 for solving differential and integral equations, linear and nonlinear has been the subject of extensive analytical and numerical studies. The method is a coupling of the traditional perturbation method and homotopy in topology. This method has a significant advantage in that it provides an approximate solution to a wide range of nonlinear problems in applied sciences.

In this paper, we apply HPM to obtain the frequency of nonlinear oscillator. The solution obtained is of high accuracy which is valid for the whole solution domain.

2.0.   **Homotopy Perturbation Method.**

We consider a general nonlinear oscillator of the form:

$$mu'' + \omega_0^2 u + kf(u, u', u'') = 0 ....(1)$$

according to [7], we expand m and ω_0^2 as follows:

$$\omega_0^2 = \omega^2 + p\omega_1 + p^2\omega_2 + ... + p^n\omega_n ....(2).$$

$$m = 1 + pm_1 + p^2 m_2 + ... + p^n m_n ....(3)$$

Where p is a homotopy parameter, ω_i and m_i are unknown constants to be further determined.

3.0.   **Mathematical Pendulum**

When friction is neglected, the differential equation governing the free oscillation of the mathematical pendulum is given by:

$$u'' + \omega^2 \sin u = 0 ....(4)$$

$$u(0) = A, u'(0) = 0.$$

where

u is the angle of deviation from the vertical equilibrium position.

$\omega^2 = \dfrac{g}{l}$; is the acceleration due to gravity and l is the length of the pendulum.

The model look simple if the approximation $\sin u \sim u$ is used, the equation (4) becomes

$$u'' + \omega^2 u = 0 ....(5)$$

To obtain more accurate result we modify the above equation by putting

$$\sin u \sim u - \frac{u^3}{6} ....(6)$$

Substituting for equation (6) in (5) we have;

$$u'' + \omega^2 u - \frac{\omega^2}{6} u^3 = 0 ....(7)$$

4.0.   **Application of HPM**

Equation (7) can be written in the form of equation (1) such that.

$$u'' + \omega_0^2 u + ku^3 = 0, k = -\frac{\omega_0^2}{6} \quad\text{...(8)}$$

Applying equation (2) in equation (8), we have;

$$u'' + (\omega^2 + pc_1)u + pku^3 = 0 \quad\text{...(9)}$$

The basic assumption is that:

$$u(t) = u_0 + pu_1 + p^2 u_2 + p^3 u_3 + ... + p^n u_n = 0 \quad\text{....................................(10)}$$

When $p = 1$ equation (9) becomes

$$u'' + (\omega^2 + c_1)u + ku^3 = 0 \quad\text{...(11)}$$

Comparing equation (8) and (11);

$$\omega_0^2 = \omega^2 + c_1 \quad\text{...(12)}$$

Substituting equation (10) in equation (9) and equating the coefficients

of like powers of p.

$$u_0'' + \omega^2 u_0 = 0, u_0(0) = A, u'(0) = 0 \quad\text{...(13)}$$

$$u_1'' + \omega^2 u_1 + c_1 u_0 + ku_0^3 = 0, u_0(0) = A, u'(0) = 0 \quad\text{..(14)}$$

The solution to equation (13) is

$$u_0 = A\cos\omega t \quad\text{...(15)}$$

Putting equation (15) in equation (14) and eliminating the secular term;

we have.

$$c_1 = -\frac{3}{4}kA^2 \quad\text{...(16).}$$

From equation (12)

$$\omega^2 = \omega_0^2 + \frac{3}{4}kA^2 \quad\text{...(17)}$$

Therefore,

$$\omega^2 = \omega_0^2(1 - \frac{1}{8}A^2) \quad\text{...(18)}$$

The period (T) is therefore:

$$T = \frac{2\pi}{\omega_0 \sqrt{1 - \frac{1}{8}A^2}} \dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots(19)$$

This compared favourably with the solution obtain in [6] and the value given in [8].
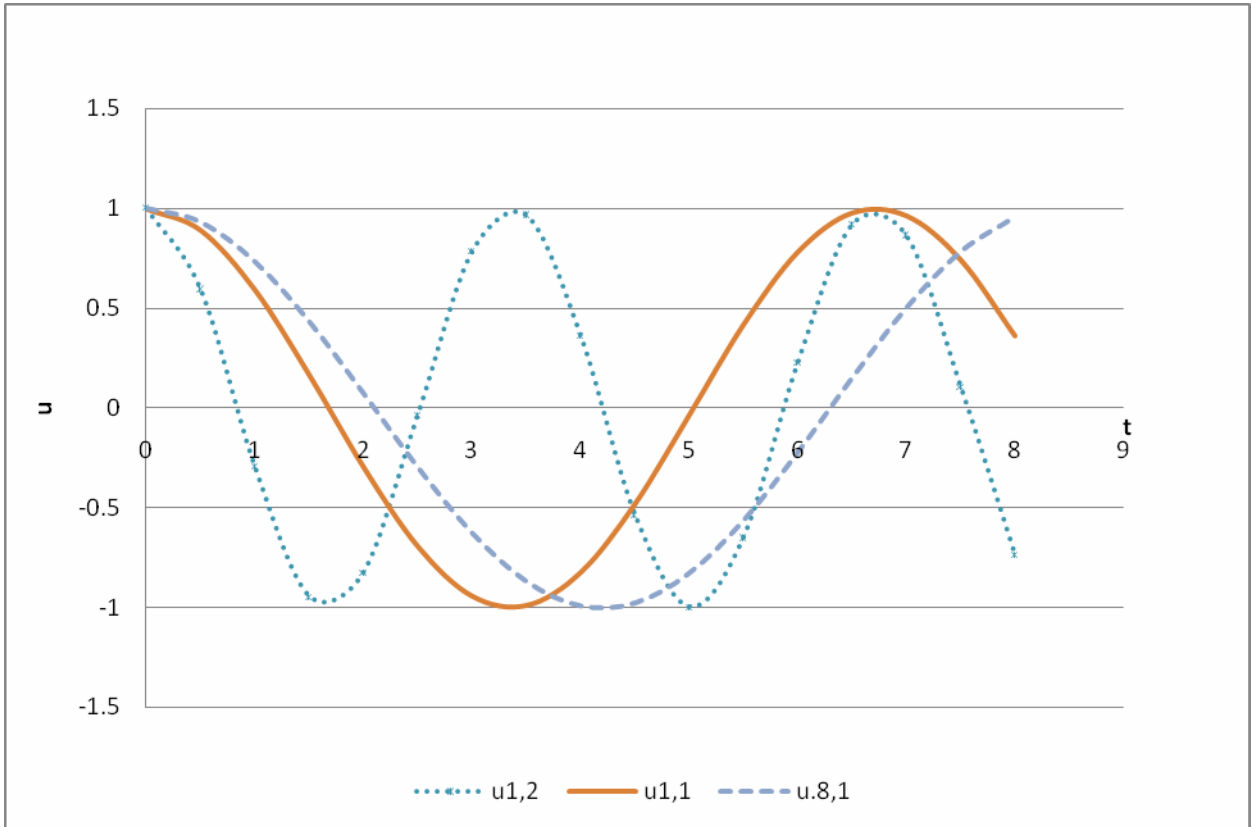
$$for\ u_{0.8,1}\ \omega_0 = 0.8, A = 1,\quad for\ u_{1,1}\ \omega_0 = 1, A = 1,\quad for\ u_{1,2}\ \omega_0 = 1,\ A = 2$$

5.0. **Conclusion**

In this work, the homotopy perturbation method has been successfully applied to find the approximate solutions for the nonlinear system.

It is worth mentioning that the method is capable of reducing the volume of computational work while still maintaining high accuracy of the numerical result. This amounts to the improvement of performance of approach. This method is relatively new and may lead to some novel and innovative applications in solving linear and nonlinear problems.

The method which proved to be a powerful mathematical tool to nonlinear oscillators can be used as a searching tool for the period or frequency of various nonlinear oscillatory systems.

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ON THE RESPONSE OF LOADED BEAM SUBJECTED TO MOVING MASSES AND EXTERNAL FORCES

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ABSTRACT: A theory describing the response of a loaded beam subjected to moving masses and external forces is considered. The governing equation is a fourth order partial differential equation. The finite Fourier sine transformation is used to transform the governing partial differential equation into second order ordinary differential equations. The ordinary differential equations for moving forces and moving masses are solved with Laplace transformation method. Number examples are used to demonstrate the efficiency of the solution. Numeric analysis shows that for a simple supported beam, the resonance frequency is lower with corresponding decrease in maximum amplitude when inertial is considered.
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Keyword: Beam, Transformation, Inertial, Resonance,
Classification: Dynamical System

1.0   INTRODUCTION

A beam or **"girder"** bridge is the simplest kind of bridge. In the past they may have taken the form of a log across a stream, but today they are more familiar to us as large box steel girder bridges. There are lots of different types of beam. A beam bridge needs to be stiff. It needs to resist twisting and bending under load. In its most basic form, a beam bridge consists of a horizontal beam that is supported at each end the pair. The weight of the beam pushes straight down on the pairs under load.

Moving loads causes solid bodies to vibrate intensively. Particularly at high velocities. Thus, the study of the behaviors of bodies subjected to moving lends has been the concern of several investigators. Among the earliest work in this area of study was the work of Timoshenko (1992) who considered the problem of simply supported Unite beams resting on an elastic Foundation and traversed by moving loads. In his analysis, he assumed that the loads were moving with constant velocities along the beam. Furthermore, Kenny (1954) took up the problem of investigating the dynamic response of infinite elastic beams on elastic foundation when the beam is under the influence of a dynamic load moving with constant speed. Lie included the effects of viscous damping in the governing differential equation of motion. More recently, Oni (1990) considered the problem of a harmonic time variable concentrated force moving at a uniform velocity over a Unite deep beam. The methods of integral transformations are used. In particular, the Unite Fourier transform is used for the length coordinate and the Laplace transform the time coordinate. Series solution, which converges as obtained for the deflection of simply supported beams. The analysis of the solution was carried out for various speeds of the load. Oni (2001) used the Galerkin method to obtain the response to several moving masses of a non-uniform beam resting on an elastic foundation. The effects of the elastic foundation on the transverse displacement of the non-uniform beam were analyzed for both the moving mass and the associated moving force problems. Awodola,T.O (2005) worked on the influence of foundation and axial force on the vibration of a simply supported thin (Bernoulli Euler) beam, resting on a uniform foundation, under the action of a variable magnitude harmonic load moving with variable velocity is investigated in the paper. The governing equation is a fourth order partial differential equation. For the solution of this problem, in the first instance, the finite Fourier sine transformation is used to reduce the equation to a second order partial differential equation. The reduced equation is then solved using the Laplace transformation. Numerical analysis shows that the transverse deflection of the thin beam, resting on a uniform foundation, under the action of a variable magnitude harmonic load moving with variable velocity decreases as the foundation constant increases. It also shows that as the axial force increases, the transverse deflection of the thin beam decreases.

Furthermore, Milormir, Stanisic .M, and Hardin, J. C. (1969) developed a theory describing the response of a Bernoulli-Euler beam under an arbitrary number of concentrated moving masses. The theory is based on the Fourier technique and shows that, for a simply supported beam, the resonance frequency is lower with no corresponding decrease in maximum amplitude when the inertia is considered.

ii**. Formulation of the problem**

The vibration of a uniformly simply supported beam carrying an arbitrary number of discrete masses m, m_2, -----m_N is considered. This mass m_t is assumed to strike the beam at t = 0 and travel across it with velocity $v_i t$. The equations of motion, with damping neglected is written as

$$\left[mA + \sum_{i=1}^{N} m_i \partial (u - v_i t) \frac{\partial^{2y}}{\partial t^2} \right] + cd \frac{\partial y}{\partial t} + EI \frac{\partial^{4y}}{\partial x^4} + Ky + \frac{\rho \partial^{2y}}{\partial x^2} = gf(x,t) \tag{1}$$

Where f (x, t)= $\sum_{i=1}^{N} m_i \partial (x - v_i t)$ (2)

EI = the flexural rigidity of the beam
f (x,t) = the transverse deflection of the beam
ρ = the mass density of the beam material
A = cross sectional area of the beam
g = the acceleration due to gravity
k = the foundation constant.
L = the length of the beam

$\partial (x - v_i t)$ is the Dirac delta function define to be zero everywhere except x=v_it, i.e $\partial (x - v_i t)$=0, $x \neq v_i t$ and

in addition $\int_0^L \partial (x - v_i t) dx = 1$ (3)

The boundary conditions are
Y (o, t) = Y (L, t) = 0
Y_{xx} (o, t) = Y_{xx} (L, t) = 0 (4)
Applying the Fourier finite sine transform, i.e

$$= \quad Z(m,t) = \int_0^L Y(x,t) Sin \frac{m\pi x}{L} dx \tag{5}$$

$$\int_0^L MA + \sum_{i=1}^{N} m_i \partial (x - v_i t) \frac{\partial^{2y}}{\partial t^2} Sin \frac{m\pi x}{L} dx + cd \int_0^L \frac{\partial y}{\partial t} Sin \frac{m\pi x}{L} dx + EI \int_0^L \frac{\partial^{Ny}}{\partial x^4} Sin \frac{m\pi x}{L} dx$$

$$+ Ky \int_0^L Y_{xx} Sin \frac{m\pi x}{L} dx + \rho \int_0^L \frac{\partial^{2y}}{\partial x^2} Sin \frac{m\pi x}{L} = \int_0^L F(x,t) Sin \frac{m\pi x}{L} dx \tag{6}$$

Solving equation (6) by integration by part, we obtained difference series solution which added together. The result becomes

$$EI \frac{m^4 \pi^4}{L^4} Z(m,t) - p \frac{m^2 \pi^2}{L^2} Z(m,t) + Kz(m,t) + MAZ_{tt}(m,t) + T \left\{ \sum_{i=1}^{N} m_i \delta(x - v_i t) \right\} Y_{tt}(m,t)$$

$$= g \sum_{i=1}^{N} m_i Sin \frac{m\pi yit}{L} \tag{7}$$

Let $Q = EI \frac{m^4 \pi^4}{L^4} - p \frac{m^2 \pi^2}{L^2} + K$ (8)

$$Qz(m,t) + mAz_{tt}(m,t) + Cz_t(m,t) + T \left\{ \sum_{i=1}^{N} m_i \delta(x - v_i t) \right\} y_{tt}(m,t)$$

$$= g \sum_{i=1}^{N} m_i Sin \frac{m\pi yit}{L} \tag{9}$$

$$Z(m,t) + \frac{mA}{Q} Z_{tt}(m,t) + \frac{C}{Q} Z_t(m,t) + \frac{T}{Q} \left\{ \sum_{i=1}^{N} m_i \delta(x - v_i t) y_{tt}(m,t) \right\}$$

$$= \frac{g}{Q} \sum_{i=1}^{N} m_i Sin \frac{m\pi v_i t}{L} \tag{10}$$

By the equation 9 we obtained the transformation equation.

$$Z_{tt}(m,t) + K_5 Z_t(m,t) + K_6 Z(m,t) = K_7 \sum_{i=1}^{N} Sin \frac{m\pi v_i t}{L} \tag{11}$$

3.0 Solution of the transformed equation.

For the purpose of the solution we consider only one mass m traveling with velocity υ. The solutions for greater numbers of masses may be obtained in the same manner .Evidently the following special cases from equation 11 follow:

(a)   Moving force: - If we neglect the inertia term, we have the classical case of a moving force. Under the above assumption equation 11 becomes

$$Z_{tt}(m,t) + K_5 Z_t(m,t) + K_6 Z(m,t) = K_7 \sum_{i=1}^{N} Sin \frac{m\pi v_i t}{L} \tag{12}$$

(b)   Moving Mass:-first approximation only the linear inertial term is considered, Equation 11 becomes.

$$Z_{tt}(m,t) + K_3 \sum_{i=1}^{N} m_i Z_{tt}(m,t) + K_5 Z_t(m,t) + K_6 Z(m,t) = K_7 \sum_{i=1}^{N} Sin \frac{m\pi v_i t}{L} \tag{13}$$

(a)   For moving force

$$Z_{tt}(m,t) + K_5 Z_t(m,t) + K_6 Z(m,t) = K_7 \sum_{i=1}^{N} m_i Sin \frac{m\pi v_i t}{L}$$

Now solving equation for moving force, we have

$$Z_{tt}(m,t) + K_5 Z_t(m,t) + K_6 Z(m,t) = 0$$

This implies that $Z(m,t) = Z_c + Z_p$ $\tag{14}$

For Z_c; equation (12) becomes

$$Z(m,t) = e^{mt}$$

$$Z_t(m,t) = Me^{mt}$$

$$Z_{tt}(m,t) = M^2 e^{mt}$$

$$\Rightarrow$$

$$M^2 e^{mt} + K_5 Me^{mt} = 0$$

$$e^{mt}(M^2 + K_5 m + K_6) = 0$$

$$e^{mt} \neq 0; then M^2 + K_5 m + K_6 = 0$$

By quadratic solution

$$M = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a} \quad a = 1, b = K_5 and c = K_6$$

$$\Rightarrow$$

$$M = \frac{-K_5 \pm \sqrt{K_5^2 - 4K_6}}{2}$$

$$M_1 = \frac{-K_5 + \sqrt{K_5^2 - 4K_6}}{2}, M_2 = \frac{-K_5 - \sqrt{K_5^2 - 4K_6}}{2}$$

Therefore

$$z_c = Ae^{m_1 t} + Be^{m_2 t}$$

$$= Ae^{\left(\frac{-K_5 + \sqrt{K_5^2 - 4K_6}}{2}\right)t} + Be^{\left(\frac{-K_5 - \sqrt{K_5^2 - 4K_6}}{2}\right)t}$$

Applying these boundary conditions
Z (m, o) = 1
Z_t (m, o) = 0
$\Rightarrow$

$$Ae^0 + Be^0 = 1 \quad or \quad A = 1 - B \tag{15}$$

$$\Rightarrow A + B = 1$$
for Z (m,o) = 0;

Let $K_7 = \left(\dfrac{-K_5 + \sqrt{K_5^2 - 4K_6}}{2} \right)^t$ and $K_8 = \left(\dfrac{-K_5 - \sqrt{K_5^2 - 4K_6}}{2} \right)^t$

$$Z(m,t) = Ae^{K_7 t} + Be^{K_8 t}$$
$$Z_t(m,t) = AK_7 e^{K_7 t} + BK_8 e^{K_8 t}$$
$$Z_t(m,o) = AK_7 e^0 + BK_8 e^0 = 0 \tag{16}$$
$$= AK_7 + BK_8 = 0$$

Sub (15) in (16)
$$(1 - B)K_7 + BK_8 = 0$$
$$K_7 - BK_7 + BK_8 = 0$$
$$BK_8 - BK_7 = -K_7$$
$$B(K_8 - K_7) = K_7$$
$$B = \frac{-K_7}{K_8 - K_7}$$

Therefore
$$A = 1 - \left(\frac{-K_7}{K_8 - K_7} \right)$$
$$= 1 + \frac{K_7}{K_8 - K_7} \quad or \quad \frac{K_8}{K_8 - K_7}$$
By expansion
$$A = \frac{K_5}{2\sqrt{K_5^2 - 4K_6}} + \frac{\sqrt{K_5^2 - 4K_6}}{2\sqrt{K_5^2 - 4K_6}}$$
$$A = \frac{K_5}{2\sqrt{K_5^2 - 4K_6}} + \frac{1}{2}$$
$$A = \frac{1}{2} + \frac{K_5}{2\sqrt{K_5^2 - 4K_6}}$$
$$B = 1 - A$$

$$\Rightarrow B = 1 - \frac{1}{2} + \frac{K_5}{2\sqrt{K_5^2 - 4K_6}}$$

$$= \frac{1}{2} + \frac{-K_5}{2\sqrt{K_5^2 - 4K_6}}$$

From $Z_c = A e^{\left(-K_5 + \sqrt{K_5^2 - 4K_6}\right)\frac{t}{2}} + B e^{\left(-K_5 - \sqrt{K_5^2 - 4K_6}\right)\frac{t}{2}}$

i.e. $Z_c = \left(\frac{1}{2} + \frac{K_5}{2\sqrt{K_5^2 - 4K_6}}\right) e^{\left(-K_5 + \sqrt{K_5^2 - 4K_6}\right)\frac{t}{2}} + \left(\frac{1}{2} - \frac{K_5}{2\sqrt{K_5^2 - 4K_6}}\right) e^{\left(-K_5 - \sqrt{K_5^2 - 4K_6}\right)\frac{t}{2}}$ \qquad (17)

For Z_p;

$$Z_p = K_g \sin \frac{m\pi v_i t}{L}$$

Where $K_g = K_4 \sum_{i=1}^{N} mi$

$$Z_p = A \frac{\sin m\pi v t}{L} + B \frac{\cos m\pi v t}{L}$$

$$Z_{tp} = \frac{A m\pi v t}{L} \cos \frac{m\pi v t}{L} - \frac{B m\pi v t}{L} Sin m\pi v t$$

$$Z_{ttp} = \frac{A m^2 \pi^2 v^2 t^2}{L^2} Sin \frac{m\pi v t}{L} - \frac{B m^2 \pi^2 v^2 t^2}{L^2} \cos m\pi v t$$

$$\Rightarrow \frac{A m^2 \pi^2 v^2 t^2}{L^2} Sin \frac{m\pi v t}{L} - \frac{B m^2 \pi^2 v^2 t^2}{L^2} \cos m\pi v t + K_5 \left(\frac{A m\pi v t}{L} \cos \frac{m\pi v t}{L} - \frac{B m\pi v t}{L} Sin m\pi v t\right)$$

$$+ K_6 \left(A \sin \frac{m\pi v t}{L} + B \cos \frac{m\pi v t}{L} = K_9 Sin \frac{m\pi v t}{L}\right)$$

$$Z = K_9 \frac{\sin m\pi v t}{L}$$

$$Z_p = A \frac{\sin m\pi v t}{L} + B \frac{\cos m\pi v t}{L}$$

$$Z_{tp} = A \frac{m\pi v t}{L} \cos \frac{m\pi v t}{L} - B \frac{m\pi v t}{L} Sin \frac{m\pi v t}{L}$$

$$Z_{ttp} = -\frac{A m^2 \pi^2 v^2 t^2}{L^2} Sin \frac{m\pi v t}{L} - \frac{B m^2 \pi^2 v^2 t^2}{L^2} \cos \frac{m\pi v t}{L}$$

The equation becomes

$$-\frac{A m^2 \pi^2 v^2 t^2}{L^2} Sin \frac{m\pi v t}{L} - \frac{B m^2 \pi^2 v^2 t^2}{L^2} \cos \frac{m\pi v t}{L} + K_5 \left(\frac{A m\pi v t}{L} \cos \frac{m\pi v t}{L} - \frac{B m\pi v t}{L} Sin \frac{m\pi v t}{L}\right)$$

$$+ K_6 \left(A \sin \frac{m\pi v t}{L} + B \cos \frac{m\pi v t}{L} = K_9 Sin \frac{m\pi v t}{L}\right)$$

$$= \left(-\frac{Am^2\pi^2v^2t^2}{L^2} - \frac{BK_5 m\pi vt}{L} - Sin\frac{m\pi vt}{L} + AK_6 Sin\frac{m\pi vt}{L} \right)$$

$$Sin\frac{m\pi vt}{L} + \left(K_5 \frac{Am\pi vt}{L} - \frac{Bm^2\pi^2v^2t^2}{L^2} + BK_6 \right) \tag{18}$$

$$cos\frac{m\pi vt}{L} + K_9 Sin\frac{m\pi vt}{L}$$

For, the solution $Z(m,t) = Z_c + Z_p$

$$Z(m,t) = \left(\frac{1}{2} + \frac{K_5}{2\sqrt{K_5^2 - 4K_6}} \right) \frac{1}{\dfrac{x - \left(-K_5 + \sqrt{K_5^2 - 4K_6}\right)}{2}} + \left(\frac{1}{2} - \frac{K_5}{2\sqrt{K_5^2 - 4K_6}} \right)$$

$$\frac{1}{\dfrac{x - \left(-K_5 - \sqrt{K_5^2 - 4K_6}\right)}{2}} + \left(\frac{K_9\left(K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}\right)}{\left(K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}\right)^2 + K_5^2 - \dfrac{m^2\pi^2v^2t^2}{L^2}} \right) \frac{\dfrac{m\pi vt}{L}}{\left(\dfrac{m\pi vt}{L}\right)^2} + \tag{20}$$

$$\left[\left(\frac{-K_9\left(K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}\right)}{\left(K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}\right)^2 + K_5^2 - \dfrac{m^2\pi^2v^2t^2}{L^2}} \right) \frac{\dfrac{K_5 m\pi vt}{L}}{K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}} \right] \frac{x}{x^2 + \left(\dfrac{m\pi vt}{L}\right)^2}$$

$$v(x,t) = \left(\frac{1}{2} + \frac{K_5}{2\sqrt{K_5^2 - 4K_6}} \right) \frac{1}{\dfrac{S - \left(-K_5 + \sqrt{K_5^2 - 4K_6}\right)}{2}} + \left(\frac{1}{2} - \frac{K_5}{2\sqrt{K_5^2 - 4K_6}} \right)$$

$$\frac{1}{\dfrac{S - \left(-K_5 - \sqrt{K_5^2 - 4K_6}\right)}{2}} + \left(\frac{K_9\left(K_6 - \dfrac{m\pi^2v^2t^2}{L^2}\right)}{\left(K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}\right)^2 + K_5 - \dfrac{m^2\pi^2v^2t^2}{L^2}} \right) \left(\frac{m\pi vt}{-L^2S^2 + m^2\pi^2v^2t^2} \right) +$$

$$\left[\left(\frac{-K_9\left(K_6 - \dfrac{m^2\Pi^2v^2t^2}{L^2}\right)}{\left(K_6 - \dfrac{m\pi^2v^2t^2}{L^2}\right)^2 + \dfrac{K_5^2 m\pi^2v^2t^2}{L^2}} \right) \frac{\dfrac{K_5 m\pi vt}{L}}{K_6 - \dfrac{m^2\pi^2v^2t^2}{L^2}} \right]$$

$$(\tag{21}$$

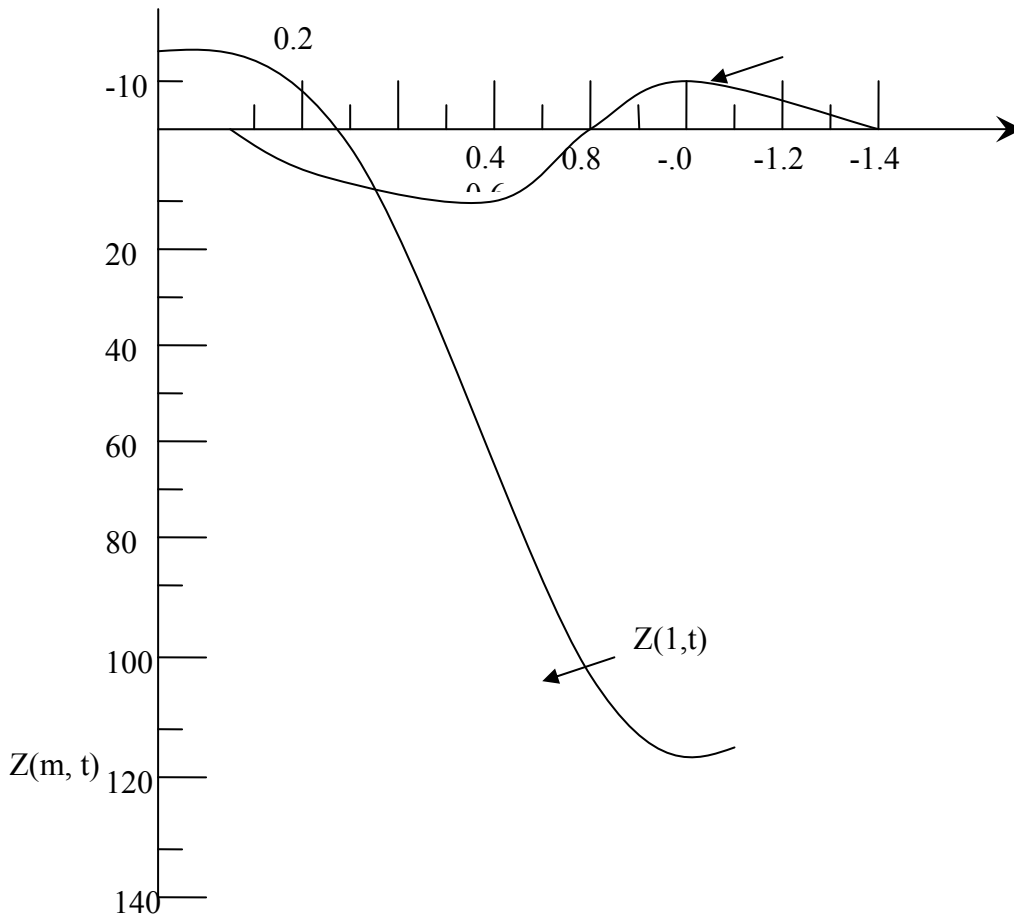Fig. 2 Convergence of coefficient for moving mass solution

By using the same method for solving the equation for moving force i.e equation (13) for moving mass becomes:-

$$Z_{tt}(m,t)\left(1+K_3\sum_{i=1}^{N}Mi\right)+K_5Z_t(m,t)+K_6Z(m,t)=K_7(m,t)=K_7\sum_{i=1}^{N}Sin\frac{m\pi vt}{L} -----(22)$$

And let $\left(1+K_3\sum_{i=1}^{N}Mi\right)=K_{10}$

$$K_{10}Z_{tt}(m,t)+K_5Z_t(m,t)+K_6Z(m,t)=K_9Sin\frac{m\pi vt}{L}$$

The solution for the moving mass becomes

$$V(m,t) = \left(\frac{1}{2} K_{10} + \frac{K_5}{2 K_{10} \sqrt{K_5^2 - 4 K_{10} K_6}} \right) \frac{1}{S - \left(- K_5 + \sqrt{K_5^2 - 4 K_{10} K_6}\right)} + \left(\frac{1}{2} - \frac{K_5}{2 K_{10} \sqrt{K_5^2 - 4 K_{10} K_6}} \right)$$

$$\frac{1}{S - \left(- K_5 - \sqrt{K_5^2 - 4 K_{10} K_6}\right)} + \left(\frac{K_9 \left(K_6 - \frac{m^2 \pi^2 v^2 t^2}{L^2} \right)}{\left(K_6 - \frac{m^2 \pi^2 v^2 t^2}{L^2} \right)^2 + K_5 \frac{m^2 \pi^2 v^2 t^2}{L^2}} \right) \left(\frac{m \pi v t}{- L^2 S^2 + m^2 \Delta^2 v^2 t^2} \right) +$$

$$\left[\left(\frac{- K_9 \left(K_6 - \frac{m^2 \pi^2 v^2 t^2}{L^2} \right)}{\left(K_6 - \frac{m^2 \pi^2 v^2 t^2}{L^2} \right)^2 + \frac{K_5^2 m^2 \pi^2 v^2 t^2}{L^2}} \right) \frac{\frac{K_5 m \pi v t}{L}}{K_6 - \frac{m^2 \pi^2 v^2 t^2}{L^2}} \right] - - - - - (23)$$

While this equation is resistant to analytic technique, it yields readily to numerical procedures. For z (m,t), the solutions for m=1,2 are tabulated in table below
Moving mass

S/N	t	Z(1,t)	Z(2,t)
1	0.0	0.000	0.000
2	0.1	0.139	0.300
3	0.2	1.078	1.726
4	0.3	3.479	4.581
5	0.4	7.785	8.025
6	0.5	14.209	9.009
7	0.6	22.766	6.817
8	0.7	33.311	1.686
9	0.8	45.559	3.321
10	0.9	59.089	7.369

Table 1.0   moving mass

Obviously higher approximations are possible by considering more terms of the series and following the same procedure. However, considering the rate of convergence of the lower-order solutions, it should not be necessary to continue this process.

4.0 Remark on the solution

In a problem such as this, one is interested in the maximum amplitude of vibration and the condition under which is can occur. For the classical solution, Eq.20, it can been shown that under certain values of the velocity the condition of resonance occurs. In this case, the amplitude of vibration becomes a linear function of time. Fig.5. the maximum value of the amplitude, which occurs when the mass is at the end of the beam.
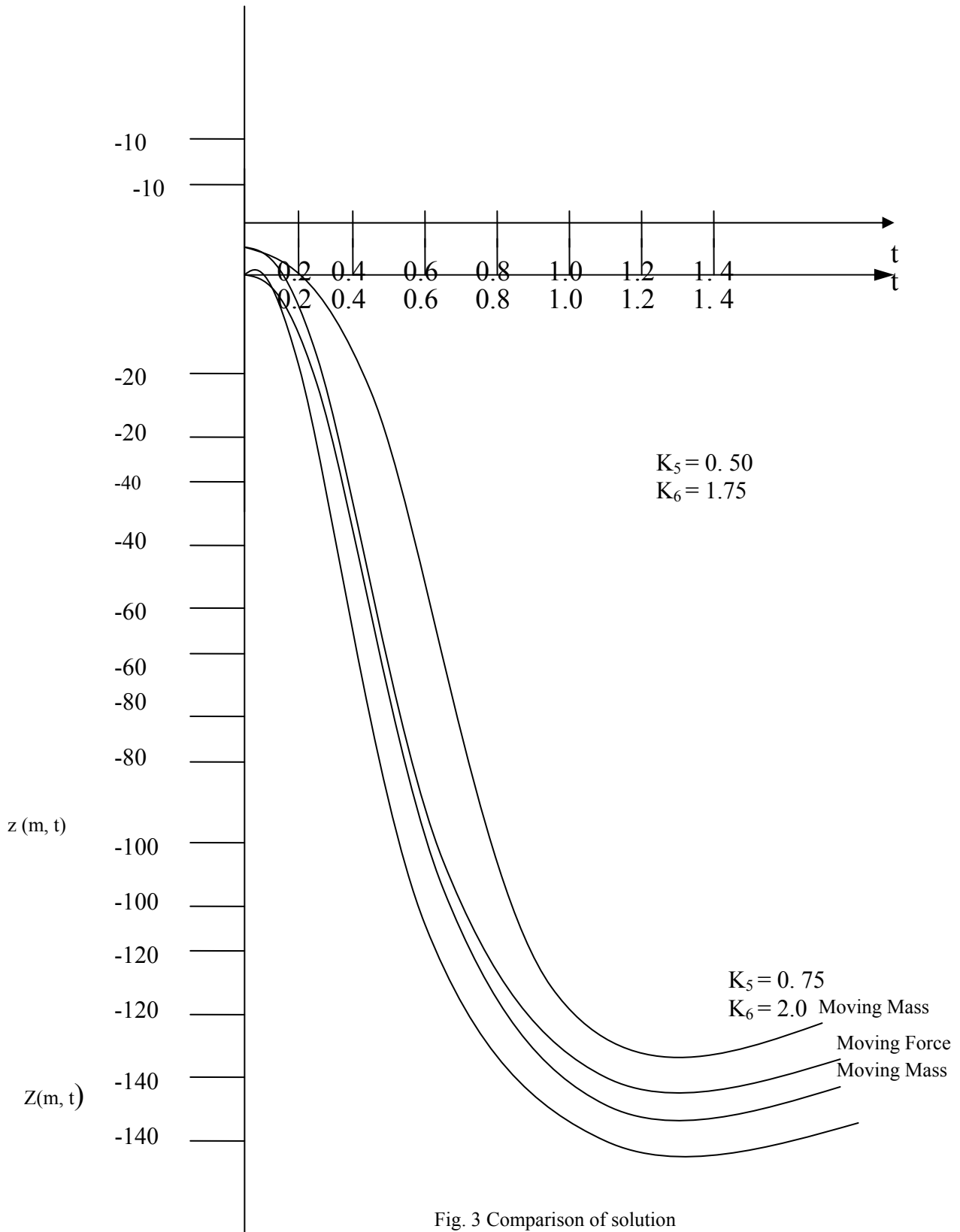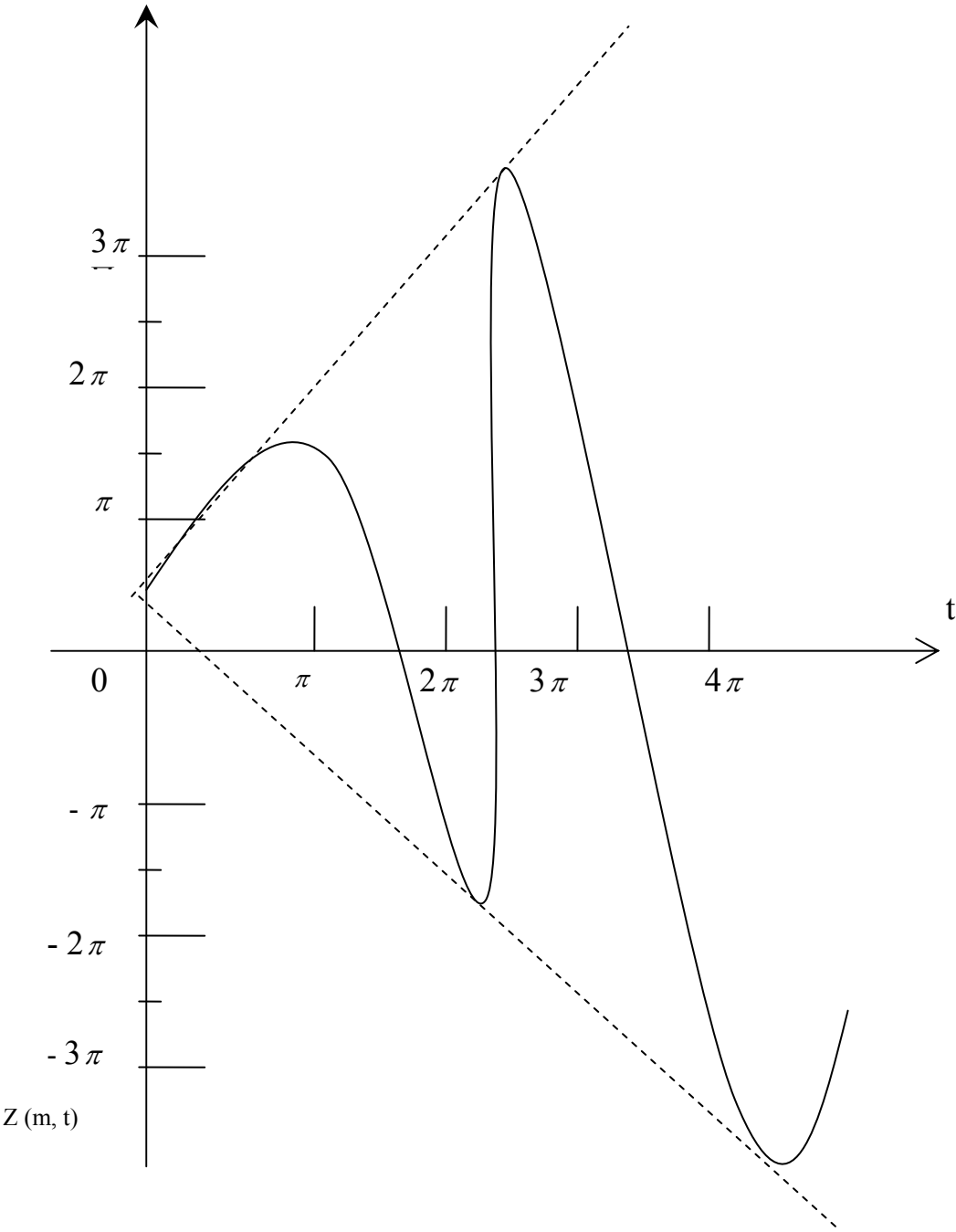
Fig. 3 Comparison of solution

Fig. 5 Amplitude growth under resonant conditions

5.0 Conclusion

A theory is presented on the response of a loaded beam subjected to moving masses and external force. The theory is simple enough to be used in computation for design considerations. The equation of motion is given in terms of δ-Dirac functions and is solved through the use of Fourier finite sine transforms. An analytic approximation is obtained and compared with the solution for a moving force. The figure five shown the amplitude of the graph which growth under a resonance conditions and figure two shown the convergence of coefficient of moving mass solution, while figures 3 and 4 shows the comparison of the solution for moving mass and moving force, which moving mass is equal to moving force solution, of It is found that, for a simply supported beam, the resonant frequency is lower with no corresponding decrease in maximum amplitude when the inertia is considered. For any higher approximation, the solution can be obtained by means of numerical techniques and for future work; the convergence of the solution can be established.

6.0   RECOMMENDATION

(a)   This work will assist the practicing engineer to evaluate the dynamic response of a loaded beam subjected to moving masses and external forces.

(b)   This work can be applied to calculations involving prestressed or reinforced beams often encountered in structural design and Construction Company.

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EVALUATION OF OXIDATIVE STRESS AND CARDIOVASCULAR DISEASE RISK FACTORS IN TYPE II DIABETIC POSTMENOPAUSAL WOMEN

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ABSTRACT

The risk of Ischemic heart disease increases in women after the menopause. Women with type 2 diabetes appear to lose the protection against cardiovascular disease afforded by estrogen. The aim of this study was to examine the relationship between oxidative stress and cardiovascular disease parameters in postmenopausal women with and without diabetes, also to evaluate the association of diabetes mellitus (DM) as a risk for coronary artery disease. This study included 50 postmenopausal women had type 2 diabetes mellitus (mean age, 57.3 ± 8.7 years) and 50 healthy postmenopausal as control (mean age, 56.9 ± 7.9 years). Oxidative stress markers, cardiovascular disease parameters and some micronutrients were measured in both groups. Our results showed that postmenopausal women with diabetes had abnormal lipid profile compared with nondiabetic women. Women with DM had significantly higher plasma levels of glucose, HbA1c, GGT and NO, LPO and lower levels of plasma antioxidant enzymes and total antioxidant capacity compared to women without DM. Also, the subjects with DM had significantly lower levels of plasma uric acid, ascorbic acid, and magnesium in comparison to those without DM. Postmenopausal women with DM had higher values of plasma iron and Cu, but the differences were not significant compared with control group. Plasma GGT was positively correlated with LDL-c level, glucose and HbA1c in postmenopausal women with DM. On other hand, plasma NO level was positively correlated with body mass index, Waist circumference, total cholesterol, triglyceride, glucose and HbA1c and negatively correlated with HDL-c, uric acid and total antioxidant capacity (TAC). Moreover, a significant inverse correlation of plasma TAC with triglyceride, glucose and HbA1c were observed. Furthermore, a significant positive correlation of plasma TAC with HDL-c and uric acid were seen too. In conclusion, there is an association between postmenopausal status, DM and cardiovascular risk parameters. This abnormality was associated with increased oxidative stress and impaired antioxidant defense in particular in type 2 diabetic patients before the development of secondary complications. These alterations contribute to the increased risk for occurrence of vascular diseases in such women.

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INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia together with biochemical alterations of glucose and lipid peroxidation. The oxidative stress in diabetes was greatly increased due to prolonged exposure to hyperglycemia and impaired of oxidant \ antioxidant equilibrium **(Ramakrishna & Jailkhani, 2008).** Diabetes decreases the gender protection against coronary heart disease in postmenopausal women **(Marra et al., 2002).** Assuming hyperglycemia as the only risk factor, women demonstrate a change in oxidative status due to an interaction between nitric oxide (NO) and uric acid production **(Pitocco et al., 2008).**

Aging is related to an increase in systemic oxidative stress. By-products of oxidative modification, such as lipid peroxidation of cellular structures are thought to play an important role in aging, atherosclerosis and late complication of diabetes mellitus. However, the rate of free radical formation has been amplified with aging and this may be at a rate that exceeds even the increased antioxidant capacity of the tissue (**Vincent et al., 2005**). As well, aging is often linked with environmental modifications lead to decreased micronutrient supplied, which is directly linked to antioxidant defense mechanisms. Regarding the main role of antioxidant micronutrients in avoiding accelerated aging, statistics linked to relationship between Oxidative stress and antioxidant status in postmenopausal women are limited (**Bureau et al., 2002**).

There is increasing experimental and clinical evidence showing that gamma-glutamyl transferase (GGT) level is associated with oxidative stress (**Simão et al., 2008**). Recent population-based epidemiological studies have shown a strong association of serum GGT activities within the reference interval with many cardiovascular disease risk factors. In addition, in prospective studies, baseline serum GGT activity predicted future diabetes, hypertension, and myocardial infarction. Among these diseases, serum GGT within the reference interval most strongly predicted incident type 2 diabetes (**Lim et al., 2007**). The objective of this study was to investigate whether postmenopausal diabetic women with short duration of disease and without complications have an altered oxidative status. Also, we evaluate the relationship of oxidative stress parameters with cardiovascular risk factors such as lipid profile and uric acid among diabetic and healthy postmenopausal women.

MATERIALS AND METHODS

A total of 100 Egyptian women $\geq$ 55 were included in this study. Demographic data was recorded for each subject using self-made questionnaire. An informed consent was obtained from all subjects before they participated in the study, which was approved by the ethical committee. They were selected from the Ain Shams University Hospitals.. The study included 2 groups of subjects; **Group I:** Postmenopausal healthy women (n=50); **Group II:** Postmenopausal women with non-insulin dependent type 2 diabetic mellitus (NIDDM) (n=50). They had not been taking any medicines other than antidiabetic pills for the past 3-5 years. The duration of diabetes ranged between 2-10 years and had no history of diabetic complications, such as nephropathy, neuropathy, ischemic heart disease (**Lau et al., 2005**). We excluded women who had anemia, those with acute or chronic liver illness, malignancy disease, hypertension, renal diseases, those who had obesity according to the Italian BMI charts (**Cacciari et al., 2002**) and those with postmenopausal hormone therapy. A woman was considered postmenopausal if she had not menstruated in the last 2 years (**Soules et al., 2001**).

Body Mass Index (BMI) was calculated as weight/height^2 (kg/m^2) (normal BMI (18.5-24.9) (**Garrow & Webster, 1985**). Waist circumference (WC) was measured while the patient standing up, at the midpoint between the bottom of the rib cage and the top of the lateral border of the iliac crest during minimal respiration. Fasting blood samples (8, 12 hours overnight) have been collected into three vacutainer tubes, one containing EDTA for measurement of blood HbA$_{1c}$ and the others containing sodium fluoride for glucose (8 hours) and lipids (12 hours) measurement. The plasma was separated by centrifugation at 3400 rpm for 10 minutes at 4°C, then subdivided into aliquots and were stored at -80°C until analysis. Plasma glucose (**Barham and Trinder, 1972**), total cholesterol (**Allain, et al., 1974**), triacylglycerols (**McGowan et al, 1973**), HDL-c (**Finley et al, 1978**) and uric acid (**Fossati et al.,1980**) concentrations were determined by enzymatic methods. LDL-c was calculated by the Friedwald formula (**Friedwald et al, 1972**). Atherogenic index was calculated from ratio of total cholesterol / HDL-c (**Wilson, et al., 1980**). Glycosylated hemoglobin (HbA$_{1c)}$) was determined in whole blood using chromatographic– Spectrophotometric methods (**Bisse, et al., 1986**).

The gamma glutamyl transferase (GGT) enzyme activity is determined using kinetic colorimetric method according to **Szasz and Persijn (1974)**. The level of plasma NO was measured spectrophotometrically as nitrite concentration after reduction of nitrate by the method described by **Bories and Bories (1995).** Plasma lipid hydroperoxide (LPO) concentration was determined using commercially available Cayman chemical LPO assay kit according to **Mihaljevic et al., (1996).**

Total antioxidant capacity (**Koracevic et al., 2001**) and antioxidant enzymes like Superoxide dismutase **(Sun et al., 1995),** Catalase **(Johansson and Bory, 1988),** glutathione peroxidase **(Paglia and Valentin, 1967**) and glutathione reductase **(Carlberg and Mannervik, 1985**) concentrations were determined using commercially available Cayman chemical assay kit.

Plasma Iron (**Henry, 1984**), copper, calcium and magnesium measurements were made by atomic absorption according to **Makino and Takaha (1981**). Plasma zinc measurement was analyzed by Inductively Coupled Plasma Mass Spectrometry (**Makino and Takahara, 1981**). Plasma ascorbic acid was assayed colorimetric by the method of **Harris and Ray, 1935**.

Statistical Analysis: Data was expressed as the mean ± S.D. Statistical analysis was performed with Statistical Package for the Social Science for Windows (SPSS, version 10.0, 1999, Chicago, IL, USA). Differences between groups were analyzed by one-way analysis of variance (ANOVA). Pearson's correlation analysis was performed to determine the relationships between nitric oxide, uric acid and other cardiovascular risk variables. *P*-value <0.05 was accepted to indicate statistical significance.

RESULTS

The basic anthropometric and clinical characteristics of the postmenopausal women with and without diabetes are shown in Table 1. The mean age ± S.D of postmenopausal women without diabetes was 56.9 ± 7.9 years; and of postmenopausal women with diabetes, 57.3 ± 8.7 years. There were significant differences between the women with and without diabetes in the levels of fasting plasma glucose and HbA1c. There was no significant difference seen in women with diabetes than in those without in the Age, BMI, Systosolic and diastolic blood pressure (Table 1). Also, our results show that postmenopausal women with diabetes had increased dyslipidemia with significant higher plasma levels of TG, LDL-c and atherogenic index (P<0.001) and lower levels of HDL-c compared with nondiabetic women (Table 2).

Table (3) shows the significantly higher levels of plasma GGT (P < 0.05), LPO (P < 0.001) and NO (P < 0.001) in women with diabetes in comparison to those without, while plasma antioxidant enzymes and total antioxidant capacity were significantly lower in diabetic postmenopausal women than healthy postmenopausal women (P<0.001). Plasma uric acid and ascorbic acid levels were significantly decreased in diabetic postmenopausal women (P < 0.001) compared to healthy postmenopausal women.

Diabetic postmenopausal women had a significantly lower plasma level of Mg than control group (P<0.001). But, no statistically significant difference (P>0.05) was found between the two groups as regards plasma iron, copper, zinc and calcium levels (Table 4)

Pearson's correlation analysis represented in Table (5) revealed that, plasma GGT was positively correlated with plasma LDL-c level (r = 0.25), glucose level (r = 0.25), HbA1c (r = 0.2) in postmenopausal women with diabetes mellitus. On other hand, plasma NO level was positively correlated with BMI (r = 0.28), WC (r = 0.31), TC (r = 0.24), TG (r = 0.52), glucose (r= 0.63) and HbA1c (r= 0.5) and negatively correlated with HDL-c (r= -0.42), uric acid (r = -0.53) and TAC (r = -0.34). Moreover, a significant inverse correlation of plasma TAC with TG(r = -0.45), glucose (r= -0.48) and HbA1c (r = -0.35)   were observed. Furthermore, a significant positive correlation of plasma TAC with HDL-c (r = 0.2) and uric acid (r = 0.37) were seen too.

Table (1): Clinical and anthropometric measurements in both groups. (Mean ±S.D).

Variables	Healthy Postmenopausal Women	Diabetic Postmenopausal Women
Subjects (n)	50	50
Age (years)	56.9 ± 7.9	57.3 ± 8.7
Duration of DM (years)		5.6 ± 3.01
Waist circumference (cm)	74.42 ± 9.8	76.7 ±8.58
BMI (Kg/m^2)	22.77 ± 1.6	23.19 ± 2.8
Systolic BP (mmHg)	122.8± 17.9	117 ± 3.7
Diastolic BP (mmHg)	77 ± 8.1	76.7± 5.6
Glucose (mg/dl)	103.7 ± 12.9	218.4 ± 52 [*]
HbA$_{1C}$ (%)	5.6 ± 1.6	9.4± 2.1*

*P < 0.001

Table (2): Lipid profile of the both studied. (Mean ±S.D).

Group Parameters	Healthy Postmenopausal Women	Diabetic Postmenopausal Women	Normal values
TC (mg/dl)	195.9± 37.9	210 ± 62.2	**<200**
TG (mg/dl)	83.9 ± 17.3	173.5± 41.6 [*]	**<150**
HDL-c (mg/dl)	52.6 ± 12.7	38.08 ± 9.5 [*]	**>50**
LDL-c (mg/dl)	129.6 ± 19.8	152.8 ± 39.7 [*]	**<100**
Atherogenic index (TC/HDL-c)	4.17 ± 1.2	5.0 ± 2.3 [*]	_

*P < 0.001

Table (3): Plasma levels of oxidative stress markers and antioxidant levels   in both studied groups (Mean±S.D.)

Groups / Parameters	Healthy Postmenopausal Women	Diabetic Postmenopausal Women
GGT (U /L)	11.22 ±2.34	15.29± 7.5*
NO (µmol/ L)	49.7± 2.32	63.44 ± 12**
LPO (µM)	12.99 ± 3.9	16.83 ± 3.55**
TAC (mmol/L)	1.88 ±0.21	1.43 ± 0.56**
SOD (U/ml)	0.79 ± 0.42	0.54 ± 0.27**
Catalase (nmol/min/ml)	10.5 ± 2.4	4.51±1.15*
Glutathione peroxidase (nmol/min/ml)	51.09 ± 14.6	38.49 ± 8.7**
Glutathione reductase (nmol/min/min)	16.7 ± 6	12.57 ± 3.6**
Uric acid (mg/dl)	5.4 ± 1.2	3.4 ± 0.82**
Ascorbic acid (mg/L)	18.27 ± 1.46	14.1 ± 1.94**

*P < 0.05
**P < 0.001

Table (4): Plasma levels of some micronutrients in both studied groups (Mean ±S.D.)

Groups / Parameters	Healthy Postmenopausal Women	Diabetic Postmenopausal Women
Iron (µg /dl)	115.4 ±38	122.4 ± 38
Cupper (mg/ L)	1.05± 0.18	1.09 ± 0.1
Zinc (µg/dl)	52.8 ± 13.5	48 ± 9.4
Calcium(mg/dl)	7.3 ± 0.22	7.2 ± 0.33
Magnesium (mg/L)	20 ± 1.54	18±1.8*

*P < 0.001

Table (5): The significant Correlation analysis between plasma GGT, NO, TAC and cardiovascular risk factors in diabetic postmenopausal women.

Variables	GGT	NO	TAC
BMI	0.06	0.28*	-0.14
WC	0.04	0.31*	0.04
Systolic BP	-0.0	-0.1	0.11

Diastolic BP	0.01	-0.04	-0.04
TC	0.19	0.24*	0.01
HDL-c	-0.06	-0.42*	0.2*
Triglycerides	0.12	0.52*	-0.45*
LDL-c	0.25*	0.15	-0.18
Glucose	0.25*	0.63*	-0.48*
HbA1c	0.2*	0.5*	-0.35*
Uric acid	-0.17	-0.53*	0.37*
GGT	-	0.18	-0.09
NO	0.18	-	-0.34*
TAC	-0.09	-0.34*	-

Correlation coefficients were calculated using Pearson's Correlation Coefficient.
* *P-value* : Correlation is significant at the 0.05 level

DISCUSSION

Oxidative stress plays a major role in the pathogenesis of type 2 diabetes mellitus. Free radicals are formed disproportionately in diabetes mellitus by glucose degradation, non-enzymatic glycation of proteins, and the subsequent oxidative degradation, which may play an important role in the development of complications in diabetic patients. The generation of free radicals may lead to lipid peroxidation and formation in several damage in diabetes mellitus (**Mahboob et al., 2005**). The present study was designed to evaluate the relationships between oxidative stress related parameters and the cardiovascular risk factors, focusing on postmenopausal women with and without diabetes mellitus.

There were highly significant increases in fasting plasma glucose and HbA1c in diabetic postmenopausal women. Higher levels indicate that they have poorly glycemic control. The data obtained were similar to those presented by other authors (**Masding et al., 2003**). In addition to this, **Jain & Lim, 2001** reported that high levels of glucose can produce permanent chemical alterations in proteins and increase lipid peroxidation in a variety of experimental models of hyperglycemia. Hyperglycemia, itself, may stimulate platelet aggregation and auto-oxidation of glucose may also lead to free radical production in diabetics. On the other hand, **Marra et al., 2002** did not find any relationship between metabolic control, as evaluated by HbA1c, antioxidant status, and markers of oxidative stress. This can be explained by the fact that HbA1c is a mean evaluation of glycemic control and reflects, only in part, glycemic fluctuations, such as postprandial hyperglycemia or disglycemia states, which may induce a pro-oxidative status and may play a significant role in the pathogenesis of diabetic complications. Moreover, **Khaw et al., 2001** suggested that HbA1c was related to total mortality in the general population, determining an increased risk even for values more than 5 %.

Lipid profiles were affected by metabolic conditions, and alterations in lipid metabolism have been implicated in atherosclerosis and coronary heart disease (CHD) (**Winder, 1994**). In the present study, TG, LDL-c and atherogenic index were significantly higher (P < 0.001) and HDL-c lower (P < 0.001) in diabetic postmenopausal women when compared to healthy women. This agrees with the findings of **Gan et al., (1999),** who demonstrated that hyperlipidemia is common in type 2 diabetes mellitus and contributed significantly to the incidence of CHD. The dyslipidemia profile includes a high TG and low HDL-c. The concentrations of LDL-c show less specific changes in diabetics, but when elevated, are an important contributor to the risk of CHD. Also, **Usoro et al., (2006)** confirmed that hormonal changes associated with menopause and age alters the lipid profile in women as evidenced by higher TC, LDL-c, atherogenic index and lower HDL-c seen in postmenopausal women. An increased triglyceride level is a common feature of diabetes mellitus (**Nayak & Roberts, 2006**). Research has suggested that this is a result of reduced action of insulin on adipocytes resulting in suppression of lipolysis.

Postmenopausal women experience more type 2 diabetes and cardiovascular diseases than their premenopausal counterparts. One hypothesis concerning the increased prevalence of type 2 diabetes and cardiovascular diseases in postmenopausal women is that it may be related to age-related changes in sex-

steroid hormones. Although sex hormones do not appear to play a primary role in the etiology of type 2 diabetes, they may be related to other metabolic factors **(Crespo et al., 2002).**

Gamma;-glutamyl transferase (GGT) has been regarded as a biomarker of hepatobiliary disease and alcohol consumption/abuse (**Whitfield, 2001).** However, GGT is elaborated by extrahepatic tissues including the kidney, epididymis, fibroblasts, lymphocytes, and lung (**Karp et al., 2001).** Accumulating experimental evidence suggests an important role for GGT in extracellular catabolism of glutathione, the principal thiol antioxidant in humans. GGT enhances the availability of cysteine to promote intracellular glutathione (GSH) resynthesis, thereby counteracting oxidant stress **(Lee et al., 2007).**

There is evidence that GGT is a potential biochemical marker for the preclinical development of atherosclerosis. GGT was found to play a role in the pathogenesis of atherosclerosis because it was detected in atheromatous plaques of carotid and coronary arteries triggering the oxidation of LDL **(Paolicchi et al., 2004**). In the present study, the plasma GGT was significantly increased (P< 0.05) in diabetic postmenopausal women compared to healthy women.

If serum GGT is a marker of oxidative stress, it might have important implications both clinically and epidemiologically because measurement of serum GGT is easy, reliable, and not expensive. Mechanisms that explain the contribution of GGT to Cardiovascular disease and mortality have not been fully elucidated. Although we observed that the relations of GGT to cardiovascular events and death remained robust after accounting for fasting glucose and components of the metabolic syndrome, it is conceivable that such adjustment incompletely accounts for hepatic insulin resistance **(Lim et al., 2004).**

Nitric oxide (NO) overproduction in diabetes has been documented in several clinical studies **(Chiarelli et al., 2000).** This appears contradictory to the abundant experimental data indicating that nitric oxide is a beneficial endothelium-derived vasodilating factor that is deficient in diabetes. NO production by the endothelium is regulated by endothelial nitric oxide synthase and may respond differently to chronic hyperglycemia than does nitric oxide produced elsewhere **(Hoeldtke, 2003)**.

Furthermore, the diabetic postmenopausal women exhibited higher activities of plasma lipid hydroperoxide (LPO) (P < 0.001) than healthy women. This indicates that oxidizability of plasma as measured by LPO was greater in diabetic women as a result of increase plasma peroxide concentration in diabetic **(Heffner et al., 1995).** Our finding is similar to that by **Nourooz zadeh et al., 1997** who found that plasma LPO level was substantially higher in type 2 diabetic patient

Uric acid has been proposed to play a pivotal role in the antioxidant defense systems in humans. The intriguing question of a role for uric acid in the free radical–scavenging processes in diabetes has received little attention. Elevated glucose levels seem to be the principal mechanism, because hyperglycemia has, at least in part, an osmotic diuretic effect, leading to an excessive excretion of uric acid **(Marra et al., 2002).** In our study, the plasma NO level was significantly increased (P < 0.001) while uric acid and vitamin C levels were significantly lower in postmenopausal women with diabetes than in healthy control. This fact, together with the positive relationship found between uric acid levels and antioxidant capacity, as shown in Table 5, confirms the importance of uric acid for the antioxidant defenses .

It is likely that uric acid is degraded or metabolized when it scavenges peroxynitrite **(Santos et al., 1999).** Accordingly, the suppression of serum uric acid occurred early, at the first patient evaluation, when there was minimal uricosuria. This indicates that direct effects of peroxynitrite excess are the most important cause of uric acid suppression, at least initially. Indirect renal effects appear to play a contributory role. Although nitric oxide overproduction has been previously reported in patients with diabetes, this is a complex and poorly understood phenomenon. Our simplified interpretation does not take into account the multiple potential mechanisms for formation of reactive oxygen species in patients with diabetes, nor did we address the possibility that eNOS may generate both Superoxide anions and nitric oxide **(Xia et al., 1998).** Nitrosative stress and oxidative stress in concert lead to peroxynitrite formation and lipid peroxidation, which synergistically compromise ATP synthesis and damage mitochondria , decrease cellular viability, and promote apoptosis. Unfortunately, hyperglycemia stimulates the synthesis of reactive oxygen intermediates in multiple tissues and subcellular locations, and it is uncertain which is the most important or suppressible with antioxidants **(Kelso et al., 2002).**

Our data indicate that oxidative stress and nitrosative stress have detectable adverse effects on peripheral nerve function within the first few years of diabetes, and therefore, it may be possible to prevent them with interventions introduced early in those patients who are unable to maintain normoglycemia. we have evidence that nitric oxide overproduction occurs in patients with poorly controlled type 2 diabetes and leads to suppressed uric acid. These metabolic changes are associated with detectable adverse effects on

peripheral nerve function even in patients who have been exposed to hyperglycemia only a few years **(Hoeldtke et al., 2002).**

Teramoto et al., (2004) have found that vitamin C administration is capable of restoring endothelial function in certain high risk groups characterized by oxidative stress, and ongoing research may establish whether this treatment can reduce cardiovascular risk in a clinical setting **(Waring et al., 2006)**. Other studies suggest that people with low vitamin C levels have higher total and harmful LDL cholesterol levels and lower beneficial HDL cholesterol levels. In a study reported in the American Journal of Clinical Nutrition, USDA researchers found that high blood levels of vitamin C were associated with high levels of HDL cholesterol in 316 women and 511 men aged from 19 to 95.11 Vitamin C also helps to protect blood fats and artery walls against oxidative damage by free radicals, and seems to have beneficial effects on clotting **(Hallfrisch et al., 1994).**

Abnormally-high levels of free radicals, lipid peroxidation and simultaneous decline in antioxidant defense mechanisms can lead to damage of cellular organelles and enzymes. These consequences of oxidative stress can promote the development of complications in diabetes mellitus patients. Antioxidant enzyme-dependent defenses play an important role in scavenging free radicals produced under oxidative stress **(Mahboob et al., 2005).**

Our studies had consistently demonstrated deficiency in the antioxidant enzymes as well as a significantly decrease in total antioxidant capacity (TAC) in diabetic postmenopausal women than in healthy women , suggestive of reduced total antioxidant defense. Previous studies are consistent with our own findings of increased oxidative stress in NIDDM **(Vincent et al., 2004)**. Also, this result agrees with the finding of **Abou-Seif & Youssef, 2004** who explained this decrease could be due to increased oxidative stress and free radical formation in diabetes mellitus which resulted in decreased antioxidant enzymes. Similarly, oxidative stress is linked to preclinical features of disease, such as vascular endothelial activation that can lead to atherosclerosis . The early increase of oxidative stress in diabetes is more pronounced in women and may account for increased cardiovascular disease in female patients (**Pansini et al., 2008**)**.**

Metals play a major catalytic role in the production of free radicals. A disruption in the homeostasis of the latter two redox-active metals is particularly significant in light of the increases in oxidative stress parameters, such as lipid peroxidation, and the oxidative damage to senile plaques and nucleic acids **(Perry et al., 2002).** There is accumulating evidence that the metabolism of several trace elements is altered in diabetes mellitus and that these nutrients might have specific roles in the pathogenesis and progress of this disease **(Kazi et al., 2008).**

As regards plasma Iron and Cu levels, high mean values were detected in postmenopausal women with type II diabetes versus the nondiabetic women, but the differences found was not significant (P < 0.05). These results are consistent with those obtained in other studies, confirming that deficiency and efficiency of some essential trace metals may play a role in the development of diabetes mellitus **(Kazi et al., 2008).** Sex related differences were also reported by **Ruiz et al., (1998)** who observed significant differences in plasma copper levels between diabetic females and non-diabetic females.

Zinc is known for its ability to block Iron-mediated production of free radicals by serving as antioxidant **(Atamna et al., 2002).** In our results, the plasma zinc level was found to be low in diabetic postmenopausal women but did not reach statistically significant level as compared to healthy control. This finding is in agreement with **Galan et al., (2005)** who observed significantly lower zinc status in the elderly population.

Magnesium is known to play an important role in carbohydrate metabolism, and its imbalance has been implicated in diabetes mellitus both as a cause and a consequence. Diabetic patients have additional risk factors for hypomagnesaemia and magnesium status. It may also play a role in the release of insulin and magnesium depletion has an atherogenic potential **(Nasri and Baradaran, 2008).**

In the present study, the plasma calcium level was approximately similar in both groups; meanwhile there was a significant decrease (P< 0.001) in the level of magnesium in diabetic postmenopausal women compared to the healthy women. This observation is in agreement with a **Guerrero et al., (2006)** who founded that diabetics had lower levels of magnesium than normal men and

women. These data confirm the results shown in other studies of significantly lower levels of serum magnesium among those with fasting glucose levels equivalent to ADA criteria for diabetes. A similar observation was made by **Afridi et al., (2008)** among nonhypertensive diabetic patients.

Most factors cause a decrease rather than an increase in trace elements concentration. Decreased concentrations are related mainly to decreased nutrition intake, intestinal uptake and altered distribution while increased concentration is reported to result from excessive homeopathic intake, industrial or environmental exposure. However, in the diabetics, ageing and increasing duration of diabetes enhances urinary loss of these elements, while lower serum zinc might be attributed to the hormonal imbalance associated with the diabetic state (**Nsonwu et al., 2006).**

The correlation analysis showed the plasma GGT, NO and TAC levels were closely related to most variables of cardiovascular risk factors. In conclusion, our data suggest that type 2 diabetic women with a short duration of disease and poor metabolic control show an early imbalance in their antioxidant capacity and augmented levels of lipid, even in the absence of complications. GGT is a potential biochemical marker for the preclinical development of atherosclerosis. Reduced uric acid levels and increased nitric acid production seem to contribute to this phenomenon, especially in diabetic women. The severe alteration of the oxidative pattern together with low antioxidant capacity detected in diabetic women may offer one possible pathogenic explanation for the higher incidence of cardiovascular complications observed in diabetics versus non diabetic women. Also, there is a strong direct relationship between oxidative stress markers and cardiovascular risk factors in diabetes mellitus.

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Thrombus Formation Elevates Tissue Factor (TF) Expression in Atherosclerotic Rabbit Serum

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Abstract: Background: Tissue factor (TF), expressed in endothelial cells and enriched in human atherosclerotic lesions, acts as a critical initiator of blood coagulation in acute coronary syndrome. TF expressed on the surface of vascular wall acts as the major procoagulant for thrombus formation. To test the hypothesis that localized thrombosis is associated with systemic inflammation, we investigated whether atherosclerotic rabbits exhibited significantly high levels of TF at the site of thrombus. **Materials and Methods:** Rabbit atherosclerosis was induced by balloon deendothelialization and feeding a high cholesterol diet (1%) for 6 months, and thrombosis was triggered by Russell viper venom and histamine. Plasminogen activator inhibitor 1 (PAI-1) levels of sections from the non-thrombosis and thrombosis areas of rabbit thoracic aorta were detected by Immunohistochemical staining method. **Results:** Sites of thrombus formation have higher level of TF expression than that of non-thrombus area. There is marked in crease in TF staining noted at the site of thrombosis. **Discussion:** Since this particular observation is also supported by increased serum C-reactive protein (CRP) levels, we propose that local event is defined by activation of CD40L connecting to macrophage CD40. This leads to local generation of tissue factor that acts as a nidus for thrombus formation. This might be a potential link between generalized systemic inflammation and localized thrombosis. [Report and Opinion. 2009;1(1):91-94]. (ISSN: 1553-9873).

Keywords: tissue factor; atherosclerosis; thrombosis; rabbit; serum

Abbreviations: CRP, C-reactive protein; PAI-1, plasminogen activator inhibitor 1; TF, tissue factor

1. Introduction

Tissue factor (TF), expressed in endothelial cells and enriched in human atherosclerotic lesions, acts as a critical initiator of blood coagulation in acute coronary syndrome (Ott, 2003). Recent study have shown that activated platelets increase TF in human monocytes (Buffon, 1999). Since TF expression in monocytes and macrophage has been shown to be induced by cross-linking processes, it is plausible that CD40L expressing platelet interaction with CD40 expressing monocytes and macrophages cross link to induce TF production from the monocytes and macrophages. CD40 is a costimulatory protein found on antigen presenting cells. CD40 binds to CD154 (CD40L) on T cells to activate the antigen presenting cell and produce a variety of downstream effects. TF expressed on the surface of vascular wall acts as the major procoagulant for thrombus formation (Golino, 2003). To test the hypothesis that localized thrombosis is associated with systemic inflammation, we investigated whether atherosclerotic rabbits exhibited significantly high levels of TF at the site of thrombus.

All the life cells in the earth have a time to live and a time to die. There are two ways in which cells die: (1) Cells are killed by injury or disease. (2) Cells suicide. Programmed cell death is also called apoptosis, which is cell suicide. Apoptosis is a mechanism by which cells undergo death to control cell proliferation or in response to DNA damage (Ma, Cherng, 2005). Inflammation is one of the common reasons that gives cells problem (disease) even die. Inflammation plays a pivotal role in atherosclerosis. In addition to being a risk marker for cardiovascular disease, much recent data suggest that CRP promotes atherogenesis via effects on monocytes and endothelial cells. The metabolic syndrome is associated with significantly elevated levels of CRP. PAI-1 is also elevated in the metabolic syndrome and in diabetes, and endothelial cells are the major source of PAI-1. According to the report by Devaraj in 2003, CRP induces PAI-1 expression and activity in human aortic endothelial cells and thus has implications for both the metabolic syndrome and atherothrombosis (Devaraj, 2003).

The rise in rabbit serum CRP and PAI-1 as early as 12 to 24 hr after thrombus-triggering may indicate a potential use as immediate to evaluate not only the long-term risk but also a more short-term risk markers of events for thrombosis if PAI-1 levels increase acutely. The time factor in CRP and PAI-1 rise could be helpful in clinical assessment of evolving cardiovascular events.

2. Materials and Methods

Rabbit atherosclerosis was induced by balloon deendothelialization and feeding a high cholesterol diet (1%) for 6 months, and thrombosis was triggered by Russell viper venom and histamine. TF levels of sections from the non-thrombosis and thrombosis areas of rabbit thoracic aorta were detected by immunohistochemical staining method.

For immunohistological analysis, rabbit aortas will be rapidly removed and frozen in liquid nitrogen followed by snap-frozee in OCT compound (Tissue-Tek) after the rabbit are killed. Cryostat sections (7 µm) of the aorta will be prepared, and air-dried 30 min at room temperature prior to washing with 0.1 M PBS followed staining with the respective antibody (Primary antibody: monoclonal antibody against rabbit tissue factor, Product #4510, American Diognostica Inc., Greenwich, CT; Secondary antibody: Rhodamine Red-X-AffiniPure F(ab')2 Frag Mouse Anti-Rat IgG (H+L), Product #212-296-168, Jackson Immuno Research Laboratories, Inc., West Grove, PA, USA, http://www.jacksonimmuno.com): As negative controls, isotype control IgG will be used. After incubation with the appropriate biotinylated, affinity-purified secondary antibodies, the sections will be incubated with alkaline phosphatase-labeled streptavidin solution and visualized using a fast red substrate kit. Quantitative analysis of atherosclerotic lesions was performed by a single observer blinded to the experiment protocol. All images will be captured by microscope equipped with camera and analyzed using Adobe Photoshop 6.0 and National Institute of Health Image 1.62 Software. For all samples, the average value for 5 locations for each animal was used for analysis. Satin dye: SlowFade® Light Antifade Kit, Product #S7461, Molecular Probes, Inc., Eugene, OR.
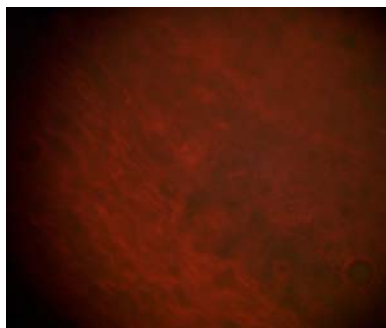
Plasminogen activator inhibitor 1 (PAI-1) levels of sections from the non-thrombosis and thrombosis areas of rabbit thoracic aorta were detected by Immunohistochemical staining method.

3. Results

It has been shown that activated platelets express CD40L and CD40L receptor, namely CD40, that are found on the surface of monocytes and macrophages interact with CD40L forming a CD40L/CD40 complex which then leads to down stream events. To test the hypothesis that localized thrombosis are associated with systemic inflammation, we investigated whether atherosclerotic rabbits which exhibited significantly high levels of serum TF at the site of thrombus. As shown in Figure 1, we have demonstrated that sites of thrombus formation have high level of TF expression. Since this particular observation is also supported by increased serum C-reactive protein (CRP) levels (Ma, 2004), we propose that local event is defined by activation of CD40L connecting to macrophage CD40. This leads to local generation of tissue factor which acts as a nidus for thrombus formation.

Sites of thrombus formation have higher level of TF expression than that of non-thrombus area. There is marked in crease in TF staining (arrow) noted at the site of thrombosis (Figure 1).

A. Without Thrombus Formation   B. With Thrombus Formation

Figure 1. Immunohistochemical staining of section from thoracic aorta of an atherosclerotic rabbit demonstrates background staining in an area without thrombus (A) compared to an area of the aorta with thrombus (B). There is marked in crease in TF staining (arrow) noted at the site of thrombosis.

PAI-1 result is described in another article.

4. Discussion

Since this particular observation is also supported by increased serum CRP levels, we propose that local event is defined by activation of CD40L connecting to macrophage CD40. This leads to local generation of tissue factor that acts as a nidus for thrombus formation. This might be a potential link between generalized systemic inflammation and localized thrombosis.

For the further study, it is important to demonstrate that systemic inflammation with rise in CRP levels stimulates CD40L activation in platelets, and enhances macrophage production of TF. Consequently, this might be a potential link between generalized systemic inflammation and localized thrombosis. Accordingly, future studies will be needed to investigate CD40L and sCD40L on platelets and TF expression in macrophages at the sites of thrombus formation by co-localization cell type specific markers and TF using immuhistochemical methods, and correlate these data to serum inflammatory markers to show the overall mechanisms underlying CRP mediated systemic inflammation developing into local thrombosis.

The Proposed mechanism of CRP mediated systemic inflammation developing into local thrombosis is shown in Figure 2.
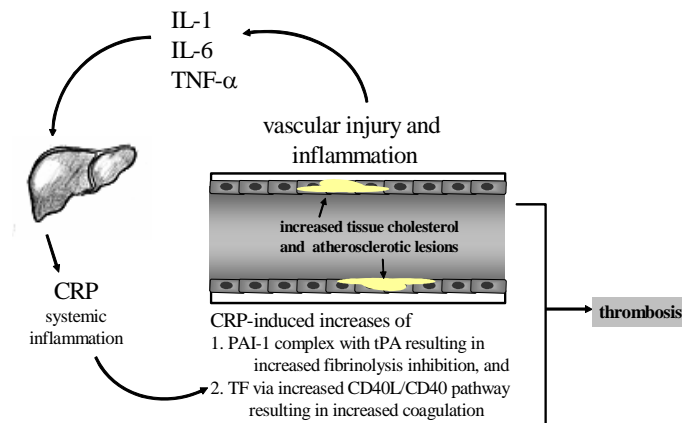
Figure 2. Proposed mechanism of CRP mediated systemic inflammation developing into local thrombosis. Administration of RVV and histamine in an atherosclerotic rabbit results in increased IL-6 (and PAI-1). IL-6 serves as a major stimulator of hepatic CRP secretion. Increased serum CRP stimulates endothelial cells (EC) synthesis and secretion of PAI-1. CRP-induced tissue factor (TF) occurs by increased CD40L-CD40 mediated activation leading to increased coagulation. Localkized TF expression by macrophages acts as a nidus for formation of thrombus at a specific site. Together with increased coagulation and fibrinolysis inhibition results in thrombosis.

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