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# Table of Content


| No. | Title | Pages |
|---|---|---|
| 01 | **Quality of Drinking Water, in Basic Schools at Almatama Locality, River Nile State, Sudan**<br>*Monzer Alzepar Saleh **Hamed**; Ali Babekier **Osman*** | 01-10 |
| 02 | **Qualitative and Quantitative Analysis of content in Ginger and its Antimicrobial, Anttioxidant Activities**<br>*Hasnaa. **Abobaker**; Hassan E. **Elkhidr**; Noor Elsham A. **Yousif*** | 11-24 |
| 03 | **Community participation & their concepts about decentralization of health programs, in Shendi locality-River Nile State, Sudan**<br>*Suleiman Elkamil Ahmed **Hamid;** Abdel Ghaffar Ali **Adam*** | 25-34 |
| 04 | ***Frequency of Staphylococcus aureus Carriage among Food Handlers in Restaurants of Atbara Market***<br>*Aliena Jaffer Ibrahim **Yousif**[1]; Waseem Sameer **Kwami**[2]* | 35-44 |
| 05 | ***Serodetection of Herpes Simplex Virus Type 2 Infection among Pregnant Women in River Nile State***<br>*Sahar Osman **Mokhtar**; Waseem Sameer **Kwami;** Salima Abdalrazig Mohammed **Alamin;** Mysoor Hassan Khalifa **Hassan*** | 45-51 |
| 06 | ***Serodetection of Helicobacter pylori* infection among gastritis patients in Atbara city**<br>*Aya Faisal Osman **Abdalrahim**, Waseem Sameer **Kwami*** | 52-60 |
| 07 | **إستخدام النماذج غير الخطية للتنبؤ بالطاقة الكهرومائية المولدة بالشركة السودانية للتوليد المائي**<br>**د.اشرف إدريس سعيد[1] ، د.محمد عبدالله موسي محمد[2]** | 61-74 |
| 08 | **Toxic effects of mercury exposure on liver function test parameters in the traditional gold mining workers in Northern Sudan.**<br>***Mansour .M. , AbedwahabAbdeen, HaghamadAllzain , Khaled. H Bakheit and Tarig . A.H. Karar*** | 75-87 |
| 09 | **On equatorial characterization of zonoids and intersection bodies**<br>*Isam Eldin **Ishag Idris*** | 88-104 |

**Research Article**

# Quality of Drinking Water, in Basic Schools at Almatama Locality, River Nile State, Sudan.

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## Abstract

Adescriptive cross-sectional school-based study was performed to evaluate the drinking water quality in basic schoolsat Almatamma locality. A questionnaire was used as a baseline survey of all basic school in the locality which is 117 in terms of school information, water quantity, quality and, sanitation facilities, sixty drinking water samples were collected from selected schools for physiochemical and bacteriological examination. Each school represent of s a group of schools that were supplied with the same water source. Conductivity, PH, TDS, Ca, Mg, Na, F, Cl, SO4, K, No3, No2, and other physiochemical parameters of water samples werewithin the permissible limits. Total iron concentration, total hardness, and degree of turbidity were above the permissible limit. The bacteriological quality of 81 % of the water samples was above the WHO and Sudanese drinking water standards. The study concluded that water, sanitation, and hygiene was poor in the basic schools at Almatama Locality, and recommended to establish rules and methods for the school WASH program's follow-up, monitoring, and evaluation, as well as encouraging community and civilian organizations to support WASH activities in basic schools.

**Keywords:** ***Drinking water quality, WASH, Basic schools, Almatamma***

## Introduction

Access to clean and inexpensive drinking water for all is one of the first targets of the Sustainable Development Goal 6 (SDG6), which was adopted in 2015(1). Access to healthy drinking water is essential to one's health (2). Unfortunately, in some parts of developing countries, where this critical resource is subject to contamination, the supply of safe drinking water has been compromised due to a lack of suitable sanitary infrastructure. Groundwater sources are being contaminated by hazardous microbiological agents and chemicals derived from a variety of social, economic, and urban activities; thus,

governmental agencies must protect groundwater sources (3, 4). Microbiological pollution has been linked to the closeness of septic tanks to artesian wells in nations with shaky basic sanitation systems, according to some studies(5,6). As a result, an estimated 2.3 billion individuals suffer from water-related illnesses around the world [7]. Drinking water contaminated with harmful microorganisms can cause major health problems related to waterborne infections [8,9]. In developing countries, drinking water is regarded as one of the most important reservoirs of bacteria. Each year, four billion instances of diarrhea are reported, with 2.2 million of them resulting in death, with children under the age of five being the most vulnerable. Furthermore, the developing world is responsible for more than 90% of the illness burden [10,11]. Each bout of diarrhea weakens the immune system, resulting in the deaths of millions more people each year [12]. The availability of improved drinking water. Adequate sanitation and a hygienic environment within schools are essential in order to provide a suitable environment to encourage school enrolment and support retention and learning achievement for children (13).Many Sudanese live in an environment characterized by poor access to improved drinking water and sanitation services. The access to improve sanitation facilities is 24% and the use of improved water sources is 55%. The situation in schools is also alarming, only 37% of the schools in Sudan have access to water, sanitation, and hygiene facilities and most of the schools with WASH facilities are in urban areas. (14).For assessing industrial, irrigation, and domestic water quality, physicochemical and microbiological characteristics are significant determinants. pH, TDS, turbidity, hardness, nitrate, EC, chloride, phosphate, and other physicochemical qualities of drinking water are among the common criteria used to present drinking water quality. If their levels are above the Sudanese drinking water standards and World Health Organization's (WHO) guideline limits. (15, 16) According to The United Nations Children's Fund, only 51% of schools in low-income settings have access to water, and 45% have adequate sanitation facilities(17). Diseases primarily transmitted through the fecal-oral route may occur through a variety of mechanisms, including consumption of contaminated water and food as well as through person-person contact (18). Flukes are one of the parasites that affected schools age group children, causing various health problems (19). Many Sudanese live in an environment characterized by poor access to improved drinking water and sanitation services.

**Material and Methods**

A descriptive cross sectional study was designed to assess the drinking water quality in basic school at Almatamma locality during 2018-2020 The consent for the study was obtained from the health and education authority The total population of the study was 117 basic schools .From data of the baseline survey the schools that have the same water sources were identified. One school was selected as represented for the group of schools of the same water source for water sample collection, hence 60 water samples were collected from the selected schools. .according to steps that set by WHO. The methods used for physicochemical parameterincluded turbidity meter for turbidity, conduct meter for conductivity. Atomic spectrophotometer for, Fe+2, titration method hardness, SO4-2, NO3-1, and F-1,The bacteriological examination included bacteriological analysis(the presence or absence of coliform, thermo tolerant coliformand E. coli . Data, were analyzed using (SPSS), and the, results were organized in tables and figures.

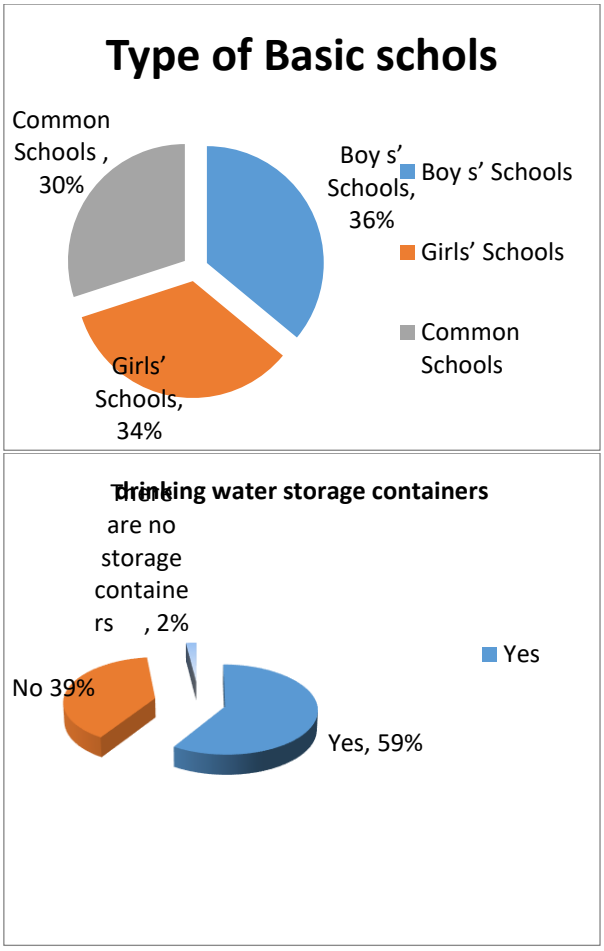



**Figure (1) Drinking water storage containers properly coveredin basic schools at Almatama Locality during 2020. {n=117}**

# Results

**Table (1) Physiochemical quality of the drinking water in basic schools at Almatmma Locality 2020. {n=60}.**

| Parameters | Explain | Frequency | Percent |
|---|---|---|---|
| Appearance | Clear | 26 | 43.3% |
| | Turbid | 34 | 56.7% |
| Remarks | Unfit | 27 | 45.0% |
| | Fit | 33 | 55.0% |
| Turbidity | Within the Standard permissible limit | 33 | 55.0% |
| | Above the Standard permissible limit | 27 | 45.0% |
| Iron | Within the Standard permissible limit | 32 | 53.3% |
| | Above the Standard permissible limit | 28 | 46.7% |
| Hardness | Moderate | 27 | 45.0% |
| | Hard | 31 | 51.7% |
| | Very hard | 2 | 3.3 % |
| Conductivity | within the Standard permissible limit | 59 | 98.3% |
| | Above the Standard permissible limit | 1 | 1.7 % |
| Total alkalinity | Within the Standard permissible limit | 34 | 56.7% |
| | Out of the Standard permissible limit | 26 | 43.3% |
| Calcium | Within the Standard permissible limit | 59 | 98.3% |
| | Out of the Standard permissible limit | 1 | 1.7 % |
| Magnesium | Within the Standard permissible limit | 25 | 41.7% |
| | Out of the Standard permissible limit | 35 | 58.3% |
| Nitrate | Within the Standard permissible limit | 59 | 98.3% |
| | Out of the Standard permissible limit | 1 | 1.7 % |
| Nitrite | Within the Standard permissible limit | 59 | 98.3% |
| | Out of the Standard permissible limit | 1 | 1.7 % |

**Cont. Table (1): Physiochemical quality of the drinking water in basic schools at Almatmma 2020. {n=60}.**

| Parameters | Explain | Frequency | Percent |
|---|---|---|---|
| Potassium | Within the Standard permissible limit | 53 | 88.3% |
| | Out of the Standard permissible limit | 7 | 11.7% |
| Fluoride | Within the Standard permissible limit | 60 | 100.0 % |
| Ammonia | Within the Standard permissible limit | 60 | 100.0 % |
| Sodium | Within the Standard permissible limit | 60 | 100.0 % |
| PH | within the Standard permissible limit | 60 | 100.0 % |
| TDS | Within the Standard permissible limit | 60 | 100.0 % |
| Chloride | Within the Standard permissible limit | 60 | 100.0 % |
| | e limit | | |
| Sulfate | Within the Standard permissible limit | 60 | 100.0 % |

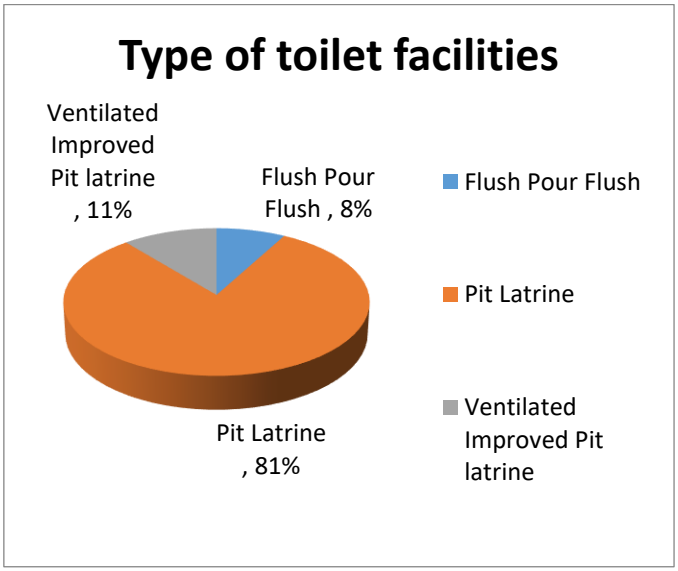


**Table (2) One Sample T test of Physiochemical analysis results of the drinking water in basic schools at Almatmma Locality 2020. {N=60}**

| Parameter | Mean | Standard deviation | T test | Sig | Test Value |
|---|---|---|---|---|---|
| Calcium | 41 | 19 | -43.2 | 0.000 | 150 |
| Calcium | 41 | 19 | -3.54 | 0.001 | 50 |
| Magnesium | 19 | 11 | -20 | 0.000 | 50 |
| Chloride | 12 | 15 | -116 | 0.000 | 250 |
| Sulfate | 18 | 18 | -96 | 0.000 | 250 |
| Nitrate | 7 | 10 | -18 | 0.000 | 33 |
| Nitrite | 0.17 | 1.2 | -10 | 0.000 | 2 |
| Fluoride | 0.23 | 0.12 | -77 | 0.000 | 1.5 |
| Ammonia | 0.07 | 0.24 | -44 | 0.000 | 1.5 |
| Sodium | 22 | 18 | -94 | 0.000 | 250 |
| Potassium | 5 | 4 | -9 | 0.000 | 10 |
| Total Iron | 11 | 24 | 3 | 0.001 | 0.3 |

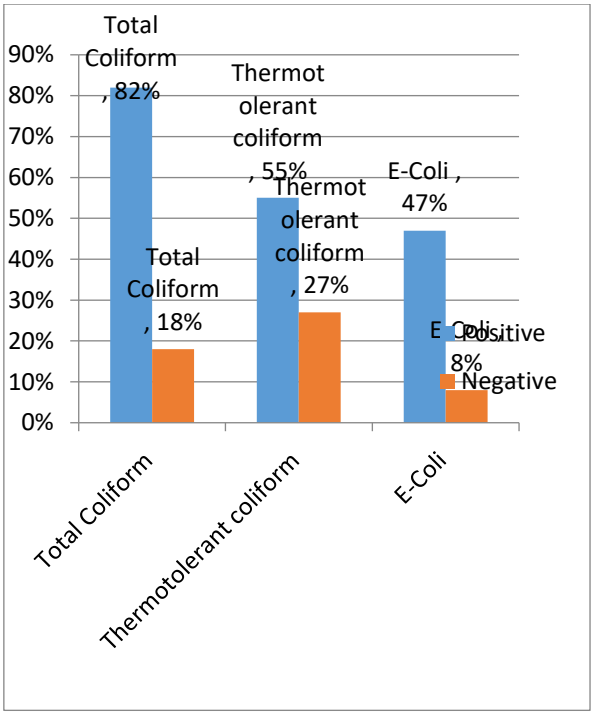



**Figure (2) Bacteriological quality of the drinking water in basic schools at Almatmma Locality 2020{n=60}**

Table (3) Correlation between the turbidity and Coliform test result of the drinking water in basic schools at Almatmma Locality 2020. {n=60}.

| Turbidity | Coliform test | | Total |
|---|---|---|---|
| | Negative | Positive | |
| Within Standard range | 18.3% | 36.7% | 55.0% |
| Above the Standard range | 0.0% | 45.0% | 45.0% |
| Total Count | 18.3% | 81.7% | 100.0% |
| X2 = 11.020 PV=0.001 significant | | | |

Table (4)Association between the turbidity and total Iron results of the drinking water in basic schools at Almatmma Locality 2020. {n=60}

| Turbidity | Total Iron | | Total |
|---|---|---|---|
| | Within the standard range | Above the standard range | |
| Within the standard range % | There are no sources in the current document.48.3% | 6.7% | 55.0% |
| Within the standard range % | 5.0% | 40.0% | 45.0% |
| Total % of | 53.3% | 46.7% | 100.0 % |
| X2 = 35.162 PV=0.000 significant | | | |

**Discussion**

The study revealed that storage containers for drinking water were present in 115(98%) of schools but in 46(39%) of schools, water containers had no proper covered, hence increased risk of contamination and spread of water-borne diseases among the student (Figure1).

The study revealed 81.7% of the water samples contaminated by coliform bacteria, 55% thermotolerant, and 46.7% of the water samples contaminated by fecal coliform E-coli (Figure2). Such findings were not incompatible with the guidelines of WHO and SSMO.Water intended for human consumption should be both safe, wholesome, and free from pathogenic agents (16). According to these results the drinking water in half basic schools were not suitable for drinking without appropriate treatment process because pupils can be exposed to the risk of the fecal-oral diseases that cause diarrhea and lead to malnutrition eventually affect the performance of the student. For the results of the physic-chemical analysis of drinking water samples, the study indicated that 45% of drinking water samples were remarked as unfit for human consumption..The results of the physic-chemical analysis of drinking water samples indicated that 45% of drinking water samples were remarked as unfit for human consumption.

Also, there are statistical differences between the standard value of WHO and SSMO (20) and analysis of the mean of Calcium, Magnesium, Chloride, Sulfate, and Potassium. The T-test value is negative which means significantvalue of the standard WHO and SSMO (PV=0,000) (Table2). For the results of the physic-chemical analysis of drinking water samples, the study indicated that 45% of drinking water samples remarked as unfit for human consumption.

There was a statistically significant relationship between the water turbidity

and coliform test (X2=11 PV=0.000), (Table 3). The turbidity can promote the growth of pathogens in the distribution system and lead to water-borne diseases. Turbidity can be indicator of a microbiological problem., There was a significant relationship between the turbidity and the result of total Iron concentration in drinking water (X2=35 and PV=0.000), (Table4).The concentration of Iron and Turbidity is high in 46%, 45% of water samples respectively(Table4) , There was a significant statistical difference between the mean of the of results(Iron and Turbidity) and values of the standard WHO, SSMO. The positive value of the T-test means the result was above the permissible limit and indicated there was a problems of the excessive total Iron concentration in water at the point of student consumption (mean= 11 T test=3 PV=0.001 test value 0.3mg/l) (Turbidity mean= 894 T-test = 4 PV= 0.000 Test value 5 NTU) (Table 1).

## Conclusion:

School'sdrinking water quality status is poor and doesn't fulfill the required standard of WHO and Sudanese Standards and Metrology Organization. The results suggest there is the problem of microbiological contamination, and schools can be a focal point for disease transmission.

Recommendations: Encourage the effort of the community and civilian organizations in support of the WASH activities in the basic schools. Public illumination and backing through broad communications, workshops, and courses on the significance and requirement for fitting financing of water sanitation and hygiene training in schools, and its importance in communicable disease control. To establish guidelines and systems to follow-up, monitoring, and evolution of the school WASH program.

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Research Article

# Qualitative and Quantitative Analysis of content in *Ginger* and its Antimicrobial, *Anttioxidant* Activities

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## Abstract

This study investigates phytochemical constituents, oil content, antimicrobial and antioxidant properties for *Ginger*. Results showed the presence of Alkaloids, Tannins, Carbohydrates, Phenols, Cardiac Glycosides, Quinones, Anthraquinone, Terpenoids, Coumarins and Steroids. Quantitative analysis for bioactive constituents in oil using GC-MS. Constituents are: Cyclohexene,3-(1,5-diethyl-4-hexenyl)-6-methylene(16.98%),1,3-Cyclohexadine,5-(1,5-dimethyl-4-hexenyl)-2-(14.94%), alpha.- franesene(10.01%), beta.-Bisabolene (7.60%), beta.-Copaene (5.06%), Benzene, 1-(1,5-dimethyl-4-hexenyl)-4-methyl-(4.98%), Eucalyptol(3.74%), Camphene(2.65%), Cyclopetene,3-isopropenyl-5,5-dimethyl-Eucalyptol(2.27%). In the disc diffustion bioassay, *Ginger* oil showed good activity against *Bacillus subtilis*, *Escherichia coli* and *Staphylococcus aureus*. Testing of antioxidant activity using 1,1-diphenyl-2-picrylhidrazil (DPPH) as free radicals, *Ginger* oil showed very high DPPH radical scavenging activity.

**Keywords: *Ginger*, antioxidant, antimicrobial, Phytochemical**

## Introduction:

Medicinal plants, since times immemorial, have been used in virtually all cultures as a source of medicine. The wide spread use of herbal remedies and health care preparations, as those described in ancient texts such as the Vedas and the Bible, and obtained from commonly used traditional herbs and medicinal plants, has been traced to the occurrence of natural products with medicinal properties [1]. Essential oils are the collection of hydrophobic secondary

metabolites that can be extracted from plants and are used in perfumes, flavorings and alternative medicine techniques such as aromatherapy. While a single compound may have a distinct smell, most essential oils are actually hundreds of compounds that, when combined, create the smell associated with that particular plant[2].*Ginger* (Zingiber officinale) is a herbaceous perennial plant of the family *Zingiberaceae*, used as a spice, food, flavouring agent, and medicine. Its generic name, Zingiber is derived from the Greek gingivitis, *Ginger* was one of the first oriental spices known in Europe. Throughout the early centuries, *Ginger* was thought to have medicinal powers. It was often used by pregnant women for morning sickness. The spice has a slightly biting taste and is used, usually dried and ground, to flavour breads, sauces, curry dishes, confections, pickles, and *Ginger* ale. Its fresh rhizome is used in cooking. It is most alkaline-promoting foods. It has a pH of 5.6 to 5.9, similar to that of figs, fennel, leeks, parsnips and romaine lettuce. The alkalinity of foods depends on many variables, including growing conditions and processing[3]. There are many studies which prove the hypotensive effect of ginger when it was given at 0.3-3 mg/kg. It helps to reduce a trial blood pressure by blocking calcium channels or by acting on muscarinic receptor[4]. *Ginger* also gave an excellent result in treating nausea. A study showed that acetone and methanol extract of *Ginger* strongly inhibits gastric ulceration. *Ginge*r oil is produced from fresh or dried rhizomes. The phytochemicals of *Ginge*r consist of volatile oils (bisabolene, cineol, phellandrene, citral, borneol, citronellol, geranial, linalool, limonene, zingiberol, zingiberene, camphene), oleoresin (gingerol, shogaol), Phenol (gingeol, zingerone), Proteolytic enzyme (zingibain), Vitamin B6, Vitamin C, Calcium, Magnesium, Phosphorus, Potassium, Linoleicacid. The pungency of *Ginger* is due to gingerol which is the alcohol group of the oleoresin [5]. The composition of fresh ginger oil shows that it contains more of oxygenated compounds (29%) compared to dryginger oil (14%). The higher content of Geranial (monoterpenoid) and other oxygenated compounds makes fresh *Ginge*r oil more potent than dry *Ginge*r oil. The content of hydrocarbon compounds are more in dry ginger oil compared to fresh ginger oil.

Additionally, zingiberol is also another predominant to 3% of volatile oils; these are bisabolene, zingiberene and zingiberol[6]. The applicability of soxhlet extraction for extraction of ginger oil by using acetone, methanol, n-hexane, water, and ethanol as solvents ~~have~~ was studied. The experimental data was presented in terms of the effect of different methods, effect of solvent, effect of time and effect of temperature on extraction of *Ginge*r oil[7]. It has also been reported that ginger (rich in sesquiterpenes) essential oils posse-ssed a wide spectrum of antimicrobial activity However, antimi- crobialactivity (bioactivity)of essential oils was dependent not only on the major components but also on the chemical structures of these components[8]. The pungent principles of ginger rhizomes, the phenolic compounds zingerone, gingerols and shogaols, are the main factors determining the antioxidant activity of the rhizome. These natural antioxidants not only inhibit lipid oxidation in food but also prevent free-radical-induced diseases caused by oxidative tissue degeneration. Oxidative stress is a major factor in the pathogenesis of diabetic complications. Mechanisms to counteract oxidative stress in tissues of living organisms are associated with the modulation of antioxidant enzymes. Several spices, among others *Ginge*r, have been proven to possess antidiabetic potential. Antioxidant activity of *Ginge*r *invivo* also influences the xenobiotic metabolism by inducing drug metabolizing enzymes such as glutathione-S-transferase, quinine reductase and glutathione peroxidase[9]. This study was aimed to determine the phytochemical constituents presence in the rhizome extract of *Ginger,* determine the yield percentage of flavonoids, Alkaloids and Saponins in the rhizome extract, analyzed oil of *Ginger* by GC-MS and study antimicrobial antioxidant activities of oil.

**Materials and methods:**

**Plant material:**

*Ginge*r from Omdurman market – Sudan. The plants seeds were blended to powder using an electric blender and stored in air-tight bottles.

**Bacterial microorganisms:**

- *Bacillus subtilis* NCTC 8236 (Gram + ve bacteria)
- *Staphylococcus aureus* ATCC 25923(Gram +ve Bacteria)

- *Escherichia coli* ATCC 25922(Gram -ve bacteria)
- *Pseudomonas aeruginosa* ATCC27853 (Gram -ve bacteria)

**Fungal microorganisms:**

A. - *Candida albicans* ATCC7596

**Preparation of extracts:**

The *Ginger* plant was washed with distilled water to remove pollutant particles, then was air dried at ambient temperature for six weeks.The dried materials were pounded using asterile laboratory mortal and grander blended using asterile electric blender to obtain a homogenous sample. 50 g of each powdered dried Rizome were extracted for 72 hours using 95% ethanol in a 200 ml beaker and later transferred to an air-tight bottle to avoid escape of the solvent. The sample was filtered using muslin cloth and later filter paper. The organic solvent filtrates were concentrated in vacuum using a rotary evaporator to obtain crude extracts. Extract obtained was kept in an air-tight bottle until it was used for phytochemical screening test.

**Phytochemical screening:**

The phytochemical screening of secondary metabolites of the plant extracts were evaluated according to the method of harborne(1973). The phytochemical screening was done to confirm the presence or absence of certain phytochemical components that are responsible for the anti-microbial activities of plant extracts such as triterpenes, carbohydrates, flavonoids, glycosides, saponins, steroids, tannins, and alkaloids[8].

**Test for Flavonoids:**

To 2ml of plant extract, 1ml of 2N sodium hydroxide was added. Presence of yellow color indicated the presence of flavonoids.

**Test for Alkaloids:**

To 2ml of plant extract, 2ml of concentrated hydrochloric acid was added. Then few drops of Mayer's reagent were added. Presence of green color or white precipitate indicated the presence of alkaloids.

**Test for Saponins**:

To 2ml of plant extract, 2ml of distilled water was added and shaken in a graduated cylinder for 15minutes lengthwise. Formation of 1cm layer of foam indicated the presence of saponins.

**Test for Tannins**:

To 1ml of plant extract, 2ml of 5% ferric chloride was added. Formation of

dark blue or greenish black indicated the presence of tannins.

**Test for carbohydrates:**

Fehlings test, Equal volume of Fehling A (copper sulphate in distilled water) and Fehling solution B (Potassium tartarate and sodium hydroxide in distilled water) reagents were mixed with few drops of crude extract and boiled, a brick red precipitate of cuprous oxide forms, indicate the presence of reducing sugar.

**Test for Phenols**:

To 1ml of the extract, 2ml of distilled water followed by few drops of 10% ferric chloride Wasadded. Formation of blue or green color indicated the presence of phenols.

**Test for Glycosides**:

To 2ml of plant extract, 3ml of choloroform and 10% ammonia solution was added. Formation of pink color indicated the presence of glycosides.

**Test for Cardiac Glycosides:**

To 0.5ml of extract, 2ml of glacial acetic acid and few drops of 5% ferric chloride were added. This was under layered with 1 ml of concentrated sulphuric acid. Formation of brown ring at the interface indicated the presence of cardiac glycosides.

**Test for Quinones**:

To 1ml of extract, 1ml of concentrated sulphuric acid was added. Formation of red color indicated the presence of quinones.

**Anthraquinone:**

To 1ml of plant extract few drops of 10% ammonia solution was added and the appearance of pink color precipitate indicated the presence of anthraquinones.

**Test for Terpenoids:**

To 0.5ml of extract, 2ml of chloroform was added and concentrated sulphuric acid was added carefully. Formation of red brown color at the interface indicated the presence ofterpenoids.

**Test for Coumarins:**

To 1 ml of extract, 1ml of 10% NaOH was added. Formation of yellow color indicated the presence of coumarins.

**Steroids and Phytosteroids:**

To 1ml of plant extract equal volume of chloroform was added and subjected with few drops of concentrated sulphuric acid where the appearance of brown ring indicated the presence of steroids and appearance of bluish brown ring indicated the presence of phytosteroids.

**Oil Extraction:**

The oil of the Rizome was extracted with the steam distillation method according to the methods of analysis used for chemical control of food of the National Institute of Health, 1996. A sample of 500 g of mature Rizome was extracted using hexane for 6 hours. The solvent was removed with a rotary evaporator and the oil was kept in the fridge at 4°C for further manipulation. The qualitative and quantitative analysis of the sample was carried out using GM/MS technique model (GC/MS-QP2010-Ultra) from japans 'Simadzu Company, with capillary column (Rtx-5ms-30m×0.25 mm×0.25µm). The sample was injected by using split mode, helium as the carrier gas passed with flow rate 1.69ml/min, the temperature program was started from 50$^0$C with rate 7$^0$C/min reaching 280$^0$C as final temperature degree, the injection port temperature was 300$^0$C, the ion source temperature was 200$^0$C and the interface temperature was 250$^0$C. The sample was analyzed using scan mode in the range of m/z 40-500 charges to ratio. Identification of components for the sample was achieved by comparing their retention times and mass fragmentation patterns with those available in the library, the National Institute of Standards and Technology (NIST). Results were recorded.

**Table (1): Chromatographic condition**

| | |
|---|---|
| Column oven temperature | 150.0°C |
| Injection temperature | 300.0°C |
| Injection mode | Split |
| Flow control mode | Linear velocity |
| Pressure | 139.3KPa |
| Total flow | 50.0ml/ min |
| Column flow | 1.54ml/sec. |
| Linear velocity | 47.2cm/sec. |
| Purge flow | 3.0ml/min. |
| Spilt ratio | - 1.0 |

**Antimicrobial test:**

**Bacterial suspensions:**

The method was employed in the standardization of the test organism[10]. One ml aliquots of a 24 hour broth

culture of the test organisms were aseptically distributed onto nutrient agar slopes and incubated at 37º C for 24 hours. The bacterial growth was harvested and washed off with 100 ml sterile normal saline, to produce a suspension containing about 108- 109 C.F.U/ ml. The suspension was stored in the refrigerator at 4° C till used. Serial dilutions of the stock suspension were made in sterile normal saline solution and 0.02 ml volumes of the appropriate dilution were transferred by micro pipette onto the surface of dried nutrient agar plates. The plates were allowed to stand for two hours at room temperature for the drops to dry and then incubated at 37 °C for 24 hours. After incubation, the number of developed colonies in each drop was counted. The average number of colonies per drop (0.02 ml) was multiplied by 50 and by the dilution factor to give the viable count of the stock suspension, expressed as the number of colony forming units per ml suspension. Each time a fresh stock suspension was prepared. All the above experimental conditions were maintained constantly so that suspensions with very close viable counts would be obtained.

**Preparation of fungal suspension:**

The fungal cultures were maintained on Sabouraud dextrose agar, incubated at 25 °C for 4 days. The fungal growth was harvested and washed with sterile normal saline and finally suspended in 100ml of sterile normal saline, and the suspended was stored in the refrigerator until used.

**Testing of antimicrobial susceptibility:**

The paper disc diffusion method was used to screen the antimicrobial activity of plant extracts and performed using Mueller Hinton agar (MHA). The experiment was carried out according to the National Committee for Clinical Laboratory Standards Guidelines (NCCLS, 1999). Bacterial suspension was diluted with sterile physiological solution to $10^8$cfu/ ml (turbidity = McFarland standard 0.5). One hundred microliters of bacterial suspension were swabbed uniformly on surface of MHA and the inoculum was allowed to dry for 5 minutes. Sterilized filter paper discs (Whatman No.1, 6 mm in diameter) were placed on the surface of the MHA and soaked with 20 µl of a solution of each plant extracts. The inoculated plates were incubated at 37 °C for 24 h in the inverted position. The diameters (mm) of the inhibition zones were measured.

**Antioxidant activity of Rhizome oil:**

DPPH radical scavenging assay:

*Ginger* is having powerful antioxidant activity due to its oil, which has protective effect on DNA damage. They have demonstrated this effect in many cell culture [4] with some modification. In 96-wells plate, the test samples were allowed to react with 2.2Di (4-tert-octylphenyl)-1-picrylhydrazyl stable free radical (DPPH) for half an hour at 37°C. The concentration of DPPH was kept as 300 µm. The extract was dissolved in DMSO (500µg/ml. concentration), while DPPH was prepared in ethanol. After incubation, decrease in absorbance was measured at 517 nm using multiplate reader spectrophotometer. Percentage radical scavenging activity by samples was determined in comparison with a DMSO treated control group and propyl gallate (PG). All tests and analysis were run in triplicate.

**Results and Discussion:**

The *Ginger* oil which are key species in Sudanese have own medicinal property. When volatile oil of these species extract was analyzed by GC-MS result indicated to a lot of Photochemical compounds was existed in *Ginger* oil. Furthermore, the oil have been assessed for Antimicrobial activity via the cup plate agar Diffusion bioassay against five standard human pathogens and Anti-oxidant activity.

**Photochemical analysis:**

Phytochemical screening of *Ginger* for various compound classes was carried out by using standard procedures as described by Harbome 1998, Sofowora 1993 and Trease and Evans 1989. Phytochemical screening of the rhizome extracts revealed that Flavonoids, Alkaloids, Saponins, Tannins, Carbohydrates, Phenols, Glycosides, Cardiac Glycosides, Quinones, Anthraquinone, Terpenoids, Coumarins and Steroids are present (Table 2).

**Table (2): phytochemical screening of *Ginger***

| Phytochemical compounds | Plant extract *Ginger* |
|---|---|
| Flavonoids | - |
| Alkaloids | + |
| Saponins | - |
| Tannins | + |
| Carbohydrates | + |
| Phenols | + |
| Glycosides | - |
| Cardiac Glycosides | + |
| Quinones | + |
| Anthraquinone | + |

| | |
|---|---|
| Terpenoids | + |
| Coumarins | + |
| Steroids | + |

+ (contain this phytochemical compound)

**GC-MS analysis of *Ginger*oil:**

The oil of *Ginger* was analyzed by GC-MS. Identification of the constituents was accomplished by consulting the MS (NIST); the observed fragmentation pattern was also interpreted .74 components The typical total ion chromatogram (TIC) is depicted in (Fig1). Different components were quantified and identified by their retention time and mass spectra and a tabulation of constituents is presented in table (3).

**Table (3): constituents *Ginger*roots oil**

| ID# | Name | Ret.Time | Area | Area% |
|---|---|---|---|---|
| | Camphene | 4.413 | 11439735 | 2.65 |
| | Cyclopentene, 3-isopropenyl-5,5-dimethyl- | 5.837 | 9810498 | 2.27 |
| | Eucalyptol | 5.883 | 16158329 | 3.74 |
| | Benzene, 1-(1,5-dimethyl-4-hexenyl)-4-methyl- | 14.528 | 21502456 | 4.98 |
| | 1,3-Cyclohexadiene, 5-(1,5-dimethyl-4-hexenyl)-2-methyl-, [S-(R*,S*)]- | 14.700 | 64547675 | 14.94 |
| | .beta.-copaene | 14.843 | 21847402 | 5.06 |
| | .alpha.-Farnesene | 14.948 | 43243848 | 10.01 |
| | .beta.-Bisabolene | 14.985 | 32820913 | 7.60 |
| | Cyclohexene, 3-(1,5-dimethyl-4-hexenyl)-6-methylene-, [S-(R*,S*)]- | 15.269 | 73334748 | 16.98 |

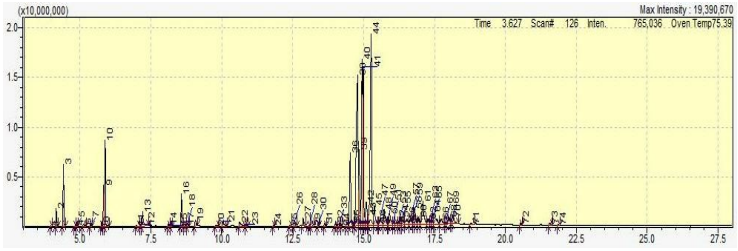


Fig.1: Total ion cromatograms of *Ginger* oil

**The following major constituents were detected by GC-MS are:**

**Cyclohexene,3-(1,5-dimethyl-4-hexenyl)-6methylene-,[S-(R*,S*)(16.98%):**

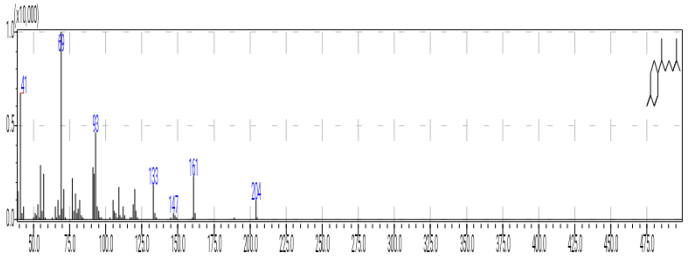


Fig.2:MassspectrumofCyclohexene,3-(1,5-dimethyl-4-hexeneyl )-6methylene-, [S-(R*,S*)]-

The El mass ofCyclohexene. In Fig. 2the peak at m/z 2o4, which appeared at R.T. 16.98 in total ion chromatogram, corresponds to $M^{+}[C_{15}H_{24}]$ The peak at m/z 189 corresponds toloss of a methyl function.

**1,3Cyclohexadine,5-(1,5-dimethyl-4-hexenyl)-2-methyl-,[S-(R*,S*)]-(14.94%):**

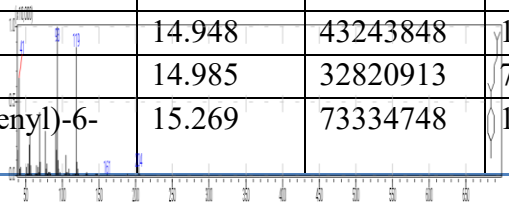


Fig.3:mass spectrum of 1,3 cyclohexadine,5-(1,5-dimethyl-4-hexenyl)-, [S-(R*,S*)]-

The El mass of Cyclohexadine. In Fig. 3 the peak at m/z 119, which appeared at R.T. 16.98 in total ion chromatogram, corresponds to $M^+[C_{15}H_{24}]^+$ The peak at m/z 105corresponds to loss of a methoxy function.

**alpha.-Farnesene (10.01%):**

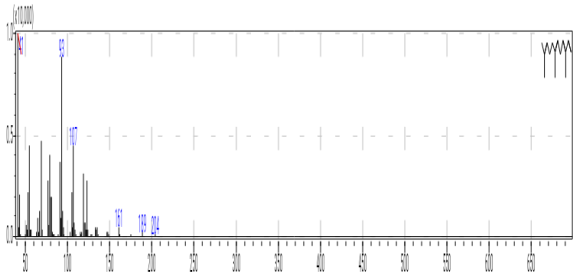


Fig.4:mass spectrum of alph.-farnesene

The El mass of alpha.- franesene. In Fig.4 the peak at m/z 204, which appeared at R.T. 14.948 in total ion chromatogram, corresponds to $M^+[C_{15}H_{24}]^+$ The peak at m/z 189 corresponds to loss of a methyl function.

**beta.- Bisabolene (7.60%):**

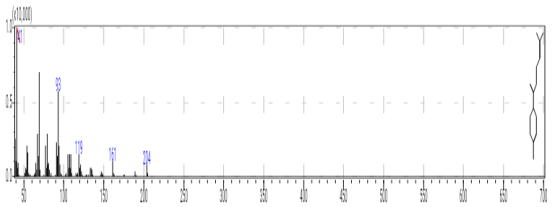


Fig.5:mass spectrum of beta.-Bisabolene

The El mass of beta.-Bisabolene. In Fig.5 the peak at m/z 204, which appeared at R.T. 14.985 in total ion chromatogram, corresponds to $M^+[C_{15}H_{24}]^+$ The peak at m/z 189corresponds to loss of a methyl function.

**beta.- copaene(5.06%):**

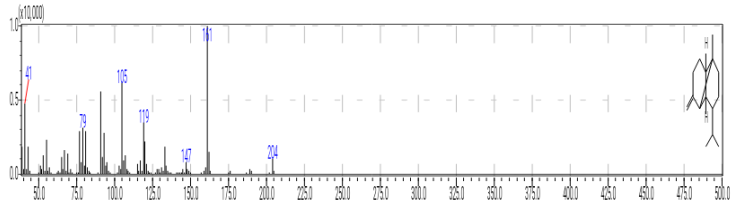


Fig. 6:mass pectrum of beta.- copaene

The El mass of beta.- Copaene. In Fig.6 the peak at m/z 204, which appeared at R.T. 14.843 in total ion chromatogram, corresponds to $M^+[C_{15}H_{24}]^+$ The peak at m/z 189corresponds to loss of a methyl function.

**Benzene, 1-(1,5-dimethyl-4-hexenyl)-4-methyl-(4.98%):**

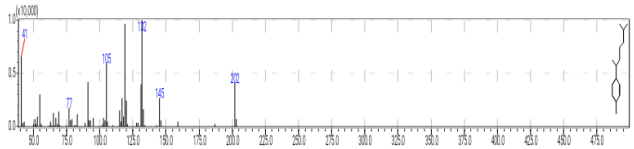


Fig.7: mass pectrum of Benzene, 1-(1,5-dimethyl-4-hexenyl)-4-methyl

The El mass of Benzene, 1-(1,5-dimethyl-4-hexenyl)-4-methyl.In Fig.7the peak at m/z 202, which appeared at R.T. 14.528 in total ion chromatogram, correspond to $M^+[C_{15}H_{22}]^+$ The peak at m/z 159corresponds to loss of a alkyl function.

**Eucalyptol (3.74%):**

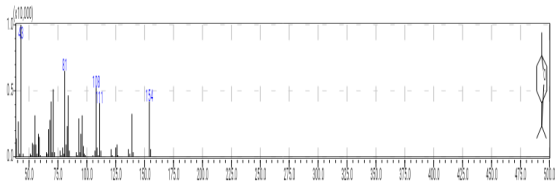


Fig.8: mass pectrum of Eucalyptol

The El mass of Eucalyptol . In Fig.8 the peak at m/z 154, which appeared at R.T. 14.5.883. in total ion chromatogram, corresponds to $M^+[C_{10}H_{18}O]^+$ The peak at m/z 139corresponds to loss of a methyl function.

**Camphene(2.65%):**

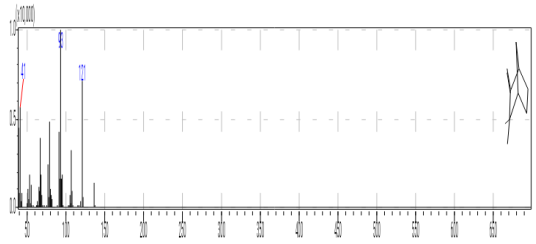


Fig.9:mass pectrum of Camphene

The El mass of camphene. In Fig. 9 the peak at m/z 136, which appeared at R.T. 4.413 in total ion chromatogram, corresponds to $M^+[C_{10}H_{16}]^+$ The peak at m/z 121 corresponds to loss of a methyl function.

**Cyclopentene, 3-isopropenyl-5,5-dimethyl-(2.27%):**

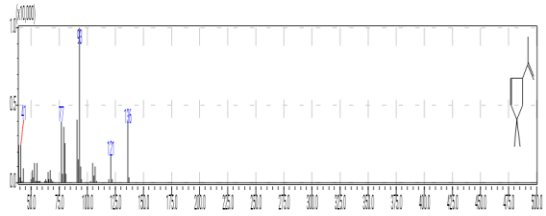


Fig.10: mass pectrum of Cyclopentene, 3-isopropenyl-5,5-dimethyl-

The El mass of in Cyclopentene, 3-isopropenyl-5,5-dimethyl- n Fig. 3.11 The peak at m/z 136, which appeared at R.T. 5.837 in total ion chromatogram, corresponds to $M^+[C_{10}H_{16}]^+$ The peak at m/z 121corresponds to loss of a methyl function.

**Antimicrobial activity:**

The target oils were evaluated for antimicrobial activity via the cup plate agar diffusion assay. The average of the diameters of the growth inhibition zones are shown in Tabls (4).The results were interpreted in terms of the commonly used terms (<9mm: inactive; 9-12 mm: partially active; 13-18mm: active; >18mm: very active). In the disc diffusion bioassay, *Giger* oil showed good activity against *Bacillus subtilis*, *Staphylococcus aureu* and *Pseudomonas aeruginosa*and Escherichia and good anticandidal potency.

**Table (4): The antimicrobial activity of *Ginger***

| Microorganism | Species | Type | Inhibition zone diameter (mm) Con (100mg/ml) |
|---|---|---|---|
| **Bacteria** | *Bacillus subtilis* | + ve | 16 |
| | *Staphylococcus aureus* | + ve | 17 |

| Microorganism | Species | Type | Inhibition zone diameter (mm) Con (100mg/ml) |
|---|---|---|---|
| | *Escherichia coli* | + ve | 15 |
| | *Pseudomonas aeruginosa* | + ve | 15 |
| **Fungi** | *Candida albicans* | + ve | 15 |

**Antioxidant activity:**

The target oils were tested for antioxidant screening for their free radical scavenging properties using 2.2Di (4-tert-octylphenyl)-1-picryl-hydrazyl (DPPH assay), while propyl galate was used as standard antioxidant. Based on findings of Dudonne, et al. (2009) [28], DPPH inhibition was categorized as very low (1-10%), low/week (10%-30%), moderate (30-60%), high (61-69%) and very high (70-100%). The Radicals scavenging assay are shown in Table (5). The oils of Ginger showed very high DPPH radical scavenging activity.

**Table 5: the radical scavenging assay (DPPH) of *Ginger*:**

| Sample | RSA% |
|---|---|
| Ginger | 85 ± 0.01 |
| propyl gallate (standard) | 92 ± 0.01 |

RSA* = Radicals scavenging assay

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**Short Notes**

# Community participation & their concepts about decentralization of health programs, in Shendi locality-River Nile State, Sudan

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## Abstract

This observational cross-sectional study was carried out in Shendi locality-River Nile State, Sudan. A sample of 653 individuals of different classes from the community, health managers, and health workers in facilities of primary health care (PHC) in the locality were selected to asses the community participation and to know their concepts about decentralization of health programs. The sample was selected using stratified sampling, then random simple sampling was used for each class. Structured questionnaires, & focus groups discussion, were used for collection of the data. (27.3%) of the community participated in health programs. In addition, there is statistical significant association between community participation in health programs and the age of the users of the health facilities. (23.3%) of the community participation is in the planning. Most of the community leaders define decentralization as delegation & distribution of responsibilities. (33.3%) of the health managers define decentralization in health system as delegation of authority. The current study shows that there is partial community participation in health programs. Health managers have to encourage this participation to be holistic and establish health programs on scientific way according to the community needs.

**Keywords:** ***Community Participation, Concepts, Decentralization, Health Programs***

## Introduction

Community is a group of people sharing common values, interests or identity, which distinguishes them from other communities, [(1)]. In addition to the health sector, families and communities need to get actively involved in taking care of their own health. Communities should participate in the following:

- Creating and preserving a healthy environment

- maintaining preventive and promotive health activities
- Sharing information about their needs and wants with higher authorities
- Implementing health care priorities and managing clinics and hospitals, [2].

Development of community participation has many concepts as community involvement, community participation, and community based initiatives (CBI). (CBI) aim to achieve all aspects of development through an integrated approach that utilizes social mobilization and empowerment to guarantee sustainable development for health. CBI strategies introduced include basic development needs (BDN), healthy villages program (HVP), healthy cities program (HCP) and women in health and development (WHD). These CBI strategies all pivot around communities playing an active role in improving the quality of life and health of the people, thus ensuring self-sufficiency and subsequently sustainable development, [3]. All that concepts go with the objectives of the millennium development goals (MDGs), which are eight international development goals that all 193 United nation member states and at least 23 international organizations have agreed to achieve by the year 2015. They include eradicating extreme poverty and hunger, achieve universal primary education, promote gender equality and empower women, reduce child mortality rates, improve maternal health, combat HIV/AIDS, malaria, and other diseases, ensure environmental sustainability, and develop a global partnership for development, [4]. The aim of the Millennium Development Goals (MDGs) is to encourage development by improving social and economic conditions in the world's poorest countries, [5]. However, the overall responsibility of the central and state Governments, the involvement of individuals, families and communities in promotion of their own health and welfare, is an essential ingredient of primary health care. Countries are now conscious of the fact that universal coverage by primary health care can't be achieved without the involvement of the local community. There must be continuing effort to secure meaningful involvement of the community in the planning. Implementation and maintenance of health services, besides maxim reliance local resources such as manpower, money and materials. In short primary health care must be built on the principle of community participation (or involvement), [6]. The promotion of primary health care, health education, immunization and environmental improvement cannot be successful unless the community participates, [7]. Community participation simply means people have to be active with the health workers and health programs, [8].

The aim of health system is health development and to achieve health for all, [9]. In

the other side to improve health status & health facilities to be accessible, decentralization in health system is needed to give more power to the local health system and to cover people by the health services. The majority of the literature define decentralization as being the process that transfers both authority and responsibility from the central government to subordinate governments, [10]. Sudan is the largest country in Africa, the large distance and continuous increasing in population need suitable means to develop and aware the community for promoting public health and improving the available health services. In addition, health system in Sudan needs decentralization to allow good coverage by primary health care.

Health in the developing world presented a gloomy picture of health, [11]. Sudan has many problems which were faced the health systems. To overcome the above obstacles, come sound of the primary health care (PHC) system adoption. In addition a new strategy came, to covered people by the health services of (PHC).

Sudan participated in WHO general assembly in 1973, and committed the strategy of PHC program in 1976, [12], [10]. Sudan health ministry suggests starting the application of the district health system in Harare in august 1987, [10], to achieve the decentralization in the health system.

Shendi locality is one of the localities of the River Nile state, which was implemented the PHC strategy and adopted the health area system, [13]. Therefore, this study done to assess the community participation & to know their concepts about decentralization of health programs.

## Methodology

This study is observational cross-sectional study. It was conducted in Shendi locality in September 2007 - April 2010, to asses the community participation and to know their concepts about decentralization of health programs. Shendi locality is one of the localities of the River Nile State. It is 173 km north of Khartoum, the capital of Sudan. The total area of the locality is about 14596 Km2, [14].

The total population of Shendi 'Mahalia' is estimated at about 245000, [15], most of them are farmers. The total number of the families are about 43340, (48.7%) of them are male and (51.3%) are female , [16]. The sample size of 653 individuals was selected of different classes from the community and health workers in the facilities of PHC in the locality by using stratified sampling then random simple sampling was used for each class of the community study as in the following:

- Total coverage of the health workers, (209).
- (30) of the health programs managers of health in ministry of health of River Nile state and in Shendi locality.
- (3o) of the community leaders.

- (384) of the users of the health facilities from the community.

Stratified sampling technique was used to select suitable sample size then a simple random sampling was run to take (384) person of the users of the health facilities from Shendi population, (245000), as a representative sample to fill the questionnaires.

A sample 384 persons were selected. This sample size was sufficient to provide 95 % assurance that the margin of error did not exceed .05 assuming that the population of Shendi locality is (245000) person. The formula used is: n = NZ2 S2 / Nd2 + Z2 S2, [8], [17].

Data was collected by the following means:

– Structured questionnaires to be filled with: Health managers, Health workers, & Users of the health facilities.

– Focus group discussion was done for: the community leaders**.**

The interviewing & groups discussion were done by the researcher him self or his trained assistants.

The collected data from the questionnaires was analyzed by entering it into computer and analyzed using the statistical package for social sciences programs (spss), and then results were presented in tables and figures. Chi-square test was used for testing the associations. The level of statistical significance was set at P-value equal to or less than 0.05 for all tests.

# Results

**Quantitative results**

**Table (1): shows the status of community participation in health programs, according to the health managers.**

**N=30**

| Variables | Frequency | Percentage |
|---|---|---|
| Participated | 17 | 56.7 |
| Not participated | 13 | 43.3 |
| Total | 30 | 100 |

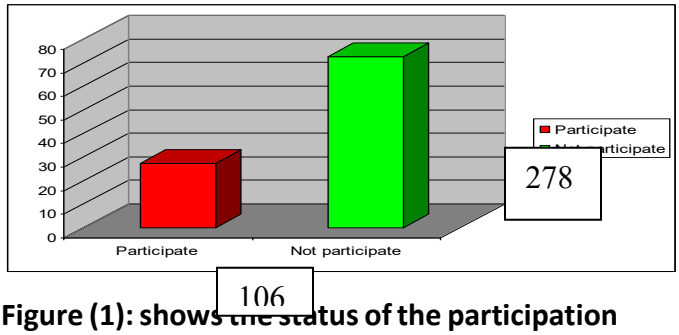



**Figure (1): shows the status of the participation of users in health programs. N=384**

(27.7%) of the users participated in health programs, and (72.3%) not participated, as in the above figure.

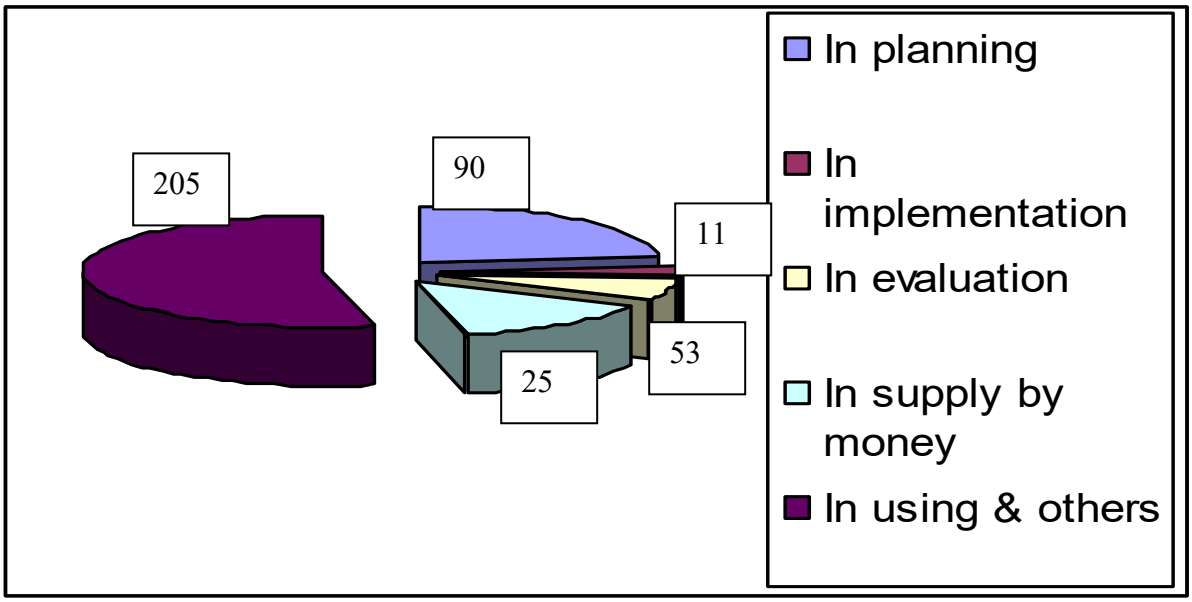



**Figure (2): shows the types of the community participation in health programs, according to the users. N=384**

(23.4%) of the community participated in planning, (3%) in implementation, (6.5%) in evaluation, (13.8%) in supply by money, (53.3%) in using of health facilities and others, as in the above figure.

**Table (2): Shows the status of the community participation according to the sex. N=384**

| Status of participation | Sex | | Total | **P-value** |
|---|---|---|---|---|
| | Male | Female | | |
| Yes | 62 | 41 | 103 | 0.053 |
| No | 137 | 144 | 281 | |
| Total | 199 | 185 | 384 | |

P-value equal or less than 0.05

No statistical significant association between community participation and the sex of the users of the health facilities, because p-value is more than 0.05.

**Table (3): shows the status of the community participation according to the age.**

**N=384**

| Status of participation | Age | | | | Total | **P-value** |
|---|---|---|---|---|---|---|
| | Less than 15 years | 15-30 years | 30-45 years | More than 45 years | | |
| Yes | 8 | 52 | 27 | 16 | 103 | 0.000 |
| No | 2 | 114 | 112 | 53 | 281 | |
| Total | 10 | 166 | 139 | 69 | 384 | |

P-value equal or less than 0.05

There is statistical significant association between community participation and the age of the users of the health facilities, because p-value is less than 0.05.

**Table (4): shows the status of the community participation according to the education. N=384**

| Participated | Education | | | | | | total | P-value |
|---|---|---|---|---|---|---|---|---|
| | Illiterate | khaloh | Primary | secondary | University | Above university | | |
| **Yes** | **14** | **7** | **26** | **23** | **32** | **1** | **103** | **0.193** |
| **No** | **42** | **14** | **70** | **94** | **59** | **2** | **281** | |
| **Total** | **56** | **21** | **96** | **117** | **91** | **3** | **384** | |

P-value equal or less than 0.05

No statistical significant association between community participation and the education of the users of the health facilities, because p-value is more than 0.05.

**Table (5): shows the status of the community participation according to the social status. N=384**

| Status of participation | Social status | | | | Total | **P-value** |
|---|---|---|---|---|---|---|
| | Single | Married | divorce | widowed | | |
| Yes | 35 | 64 | 2 | 2 | 103 | 0.309 |
| No | 79 | 177 | 17 | 8 | 281 | |
| Total | 114 | 241 | 19 | 10 | 379 | |

P-value equal or less than 0.05

No statistical significant association between community participation and the social status of the users of the health facilities, because p-value is more than 0.05.

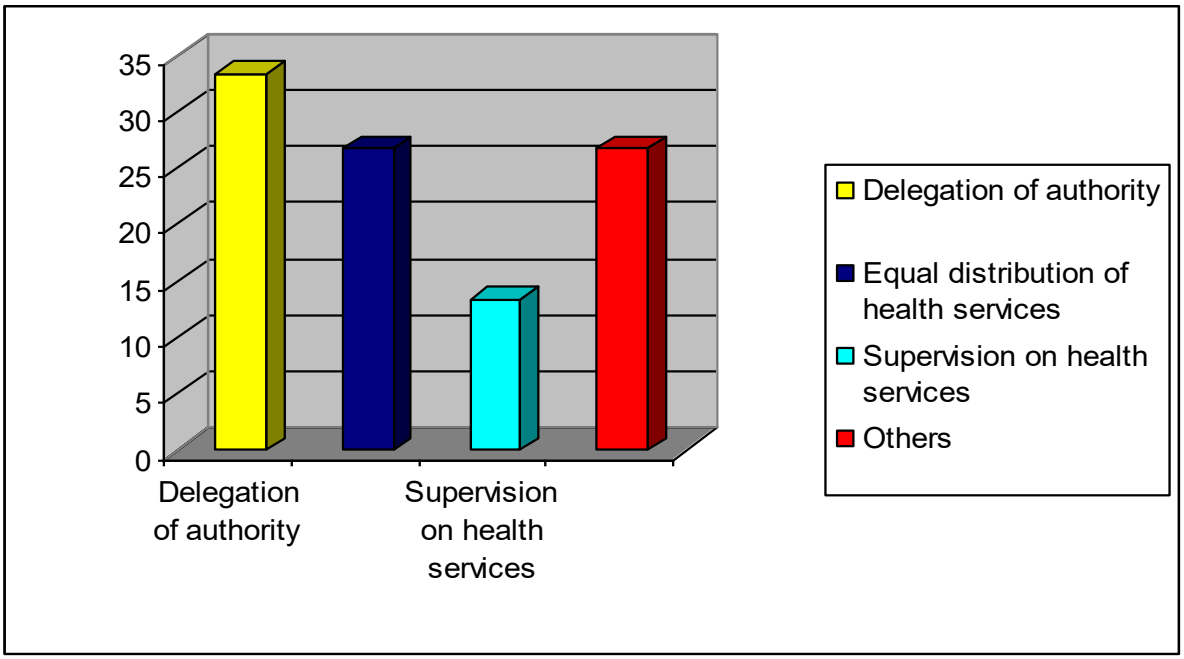



**Figure (3): shows the concepts of the health managers about decentralization - N=30**

Health leaders define decentralization in health system as, (33.3%) delegation of authority, (26.7%) equal distribution of the health services, (13.3%) supervision on health services, and (26.7%) others, as in the above figure.

## Qualitative results

**Results of focus group discussion of the community leaders**

- Most of the community leaders define decentralization, as a distribution of responsibilities.
- Health authority delegates responsibilities from up level to lower one.
- Community leaders are organizing community by dividing it to committees.
- Health programs and health problems solution come from the community.
- Health managers participate with community leaders in, planning, implementation and evaluation of the health programs.
- Their assessment of public support of the health facilities is poor.

# Discussion

Although (23.3%) of the community participated in health programs, figure (2), (72.7%) of them are not, figure (1), this result disagrees with, [6], [18], [19], & [20], "in addition to the health sector, families and communities need to get actively involved in taking care of their own health. There must be continuous effort to secure meaningful involvement of the

community in the planning, implementation and maintenance of health services. In short primary health care must be built on the principle of community participation or involvement".

No statistical significant relation between community participation and sex, education & social status of the users of the health facilities, because P-value is more than 0.05, tables (2), (4), and (5). However, the study found statistical significant relation between community participation and the age, because P-value is less than 0.05, table (3).

The concept about decentralization of the community leaders & health leaders is agreed with, [10], (the process that transfers both authority and responsibility from the central government to subordinate governments).

## Conclusion

An observational cross-sectional study was carried out in Shendi locality-River Nile State, Sudan in September 2007 - April 2010, to asses the community participation & to know their concepts about decentralization of health programs. Finally, the conclusions of this study are the following:

- (27.3%) of the users of the health facility participate in health programs.
- There is statistical significant association between community participation & age of the users of the health facilities.
- (23.3%) of the community participation is in planning.
- Most of the community leaders define decentralization as delegation & distribution of responsibilities.
- (33.3%) of the health managers define decentralization in health system as delegation of authority.

## Recommendations

Managers of health system to distribute equitably, cover people in the locality by the health facilities, & health facilities to be accessible, they have establish health programs on scientific way and on real studies according to the community needs & the size of the population they have to encourage participation of people in health programs, so to improve the health of the community.

**Acknowledgments**

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Research Article

# Frequency of *Staphylococcus aureus* Carriage among Food Handlers in Restaurants of Atbara Market

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## Abstract:

**Introduction:**The health status of the food handlers, their personal hygiene, knowledge and practice of food hygiene guidelines may play an important role in control of faeces-to-hand to-mouth spread of potentially pathogenic transient microorganisms.the objective of this study was to determine the frequency of *Staphylococcal aureus* carriage among food handlers in Restaurants of Atbara market.**Method:** A total of 50 hand swabs and 50 nasal swabs, were collected from food handlers, and then inoculated in Blood agar and MacConkeyagarmedia, incubated at 37c for 24 hours. *Staphylococcus aureus*were identified according to their colonial morphology, indirect gram staining reaction, and biochemical tests.**Results:** The study revealed that 29(58%) of food handlers were carrying *S.aureus* in their nostrils, while only 19(38%) were carrying *S. aureus* in their hands. The study denoted that there was no significant statistical association between education level, job position, and carriage of *Staphylococcus* on nose or hand, but the study found that there was significant statistical association with service year and carriage of *Staphylococcus* on hand.The study recommended that future studies should be done to determine frequency of others food borne pathogen like *Salmonella, Shiglla, E.colli*, *Listeria*, *Campylobacter*, *Bacillus* and *Clostridia species.*

**Keywords*: Atbara Market, Frequency, Food Handlers, Staphylococcus aureus Carriag***

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## Introduction

Food handler is a person who works for a food company and handles food,

regardless whether preparing or serving it. They could transmit microorganisms to the food through their skin, nasal secretions, and bowel, also from the contaminated food prepared or served by them.[1]

Foods borne disease have a dramatic impact in both developing and developed countries. The Food workers health status, their personal hygiene, knowledge and practice of food hygiene play an important role in food contamination.Food workers hands are important medium for food cross-contamination so that improved personal hygiene and hand washing would lead to the fundamental control of fecal-to-hand to-mouth spread of potentially pathogenic transient microorganisms.[1]

*Staphylococcus aureus*, *Escherichia coli* survive for hours or even days after initial contact with the microorganisms on hands and surfaces. These microbes have been associated with food borne illness for decades, causing disease and even death to many people each year, an immeasurable economic cost and human suffering.[1]

The Centers for Disease Control and Prevention has diagnosed more than400 food related illnesses. About two thirds than 400food borne illnesses one of third of all outbreaks involve bacteria. The diseases are caused either by the microorganisms themselves or by the toxins they release. [2]

Food becomes unsafe when it was contaminated with harmful organisms and/or chemical ingredients, unsafe food generates a vicious cycle causing food-borne diseases ranging from self-limiting or mild gastrointestinal symptoms (such as abdominal pain, mild diarrhea) to severe form of diseases like illness such as severe diarrhea, dysentery or cancers. [3]

Often unhealthy food handlers who have not receive appropriate health inspection can be sources of food-borne diseases to their consumers.[4]

The consumption of foods contaminated by food borne pathogenic microorganisms and the toxin they produce cause's deaths, illnesses, hospitalization, and economic losses. Due to their widespread nature, food borne diseases, in particular gastro-intestinal infections represent a very large group of pathologies with a major negative impact on public health.[2]

Many cases food-borne illness are reported every year in Africa. Numerous factors, contribute to this high number of incidents.[2]

However, it is extremely important to note that most cases offood-borne illness in the region are not reported, so the true extent ofthe problem is unknown. In most countries of the region, the surveillance infrastructure for food-borne diseases of both microbiological and chemical etiology is weak or non-existent.

This absence of reliable data on the burden of food-borne illness Impede understanding about its public health importance and prevents the development of risk based solutions to its management.[2]

In study conducted in South Africa by Lues and Tonder (2005), sample collected from hands and aprons of food handlers, the study revealed that *Staphylococcus aureus* was isolated from 88% of the population. [5]

In other study carried out in Saudi Arabia by Mushtag Ahmed Khan in (2018), 50 food handlers were screened for presence of bacteria, single non repetitive hand swab, nasal swab and swab from any wound were collected from each individual for screening, *Staphylococcus aureus* was isolated from 7.5% of the study population.[6]

In study conducted in Myanmar by Aung A, (2020). The study found that the major contaminant pathogen of hand (42.3%) and nose (54.9%) was *Staphylococcus aureus*. [7]

In Sudan Study conducted by and Hamid, 2010), who denoted that the rate of *Staphylococcus aureus* carriage was56( 21.6%) of 259 food handler. [8]

In Ethiopia, the study conducted by Assefa*et al*, (2007). The study found that there are no significant statistical association was found between bacterial contamination and (gender, education level, medical checkup, training status, hand washing habit and finger nail status) of the food handler, however there was significant association between bacterial hand contamination and service years.[2]

There are no enough studies conducted in Sudan to determine the frequency of food handler's carriage of possible contaminating pathogens which can threat human health. This study aimed to isolate and identify *staphylococcus aureus strains* which carried by food handlers work in different cafeterias of Atbara market.

**Method:**

**Collection of specimens**

A total of 50 nasal swabs, 50 swabs from right hands and left hands were collected. Nasal swab was collected aseptically from food handlers' nostrils rolling several times by applicator stick tipped with cotton and moistened with sterile normal saline. Hand swab was collected by rolling several times inside and outside the hand, between fingers and under nail from right and left hand using separate applicator stick tipped with cotton and moistened with sterile normal saline swabs were labeled with patients name and age. All specimens were transferred during two hour to the laboratory.

**Culture of specimens**

All samples were cultured in Blood agar and MacConkey agar medium, incubated at 37 c for24hrs.

**Identification of *Staphylococcus aureus***

**Colonial morphology**

The growing colonies were morphologically examined for size, color, shape, and

hemolysis on blood agar by observation of the zone around colony, clear zone alpha hemolysis, green zone beta hemolysis in blood agar media, and the colonies were examined on MacConkey for lactose fermentation, pink color indicate lactose fermentation, and yellow color indicate non lactose fermentation .

**Indirect gram stain**

Dry smear is prepared by emulsifying a colony in a drop of physiological saline and spread evenly in clean dry slide, then allowed to dry, and then the smear was fixed by passing over the flame for seconds.

Crystal violet was added to cover fixed smear for one minute, then washed by tab water, lugol's iodine was added for one minute and washed off by tab water, then decolorized by using acid alcohol for 15-20 seconds and also washed by tab water, finally safranin was added for 2 minutes and washed off by tab water then wiped the back of slide, let to dry and examined under microscope by oil immersion lens (x100).[9]

**Biochemical tests**

**Catalase test**

2ml of 3% hydrogen peroxide was transfer into sterile test tube and by using wooden stick apportion from growth of organism under test was added so release of air bubbles indicate positive result, no air bubbles indicate negative result. Positive results appear as formation of air bubbles but in negative result no air bubbles are formed.[9]

**DNase test**

By using of sterile straight loop under aseptic condition the organism under test was inoculated in the DNase agar plate and making heavy spot, the plate were incubated at 37 °C for overnight at incubator. In the end of incubation period the plate cover with hydrochloric acid, the presence of clear zone around the spot indicates positive result.[9]

**Coagulasetest**

This test used to differentiate between S.aureus (positive) from other Staphylococci (negative) the test was performed by emulsifying portion of colonies from pure growth in a drop of undiluted plasma then formation of clots in positive strains.[9]

**RESULTS**

A total of (50) samples of nasal and (50) hand (right and left) swabs were collected from (50) food handlers work in different Restaurants of Atbara market.In this study the participants age ranged from (12) to (60) years, most of them (**46%**) were within the age group (20-40years) **(Table.1).**

**Table (1): The distribution of study population according to age**

| Age (years) | No | Frequency |
|---|---|---|
| Less than 20 | 19 | 38% |
| 20—40 | 23 | 46% |
| More than 40 | 8 | 16% |
| Total | 50 | 100% |

Most of the study population (84%) in this study they do not complete their study and just

reach the elementary and secondary levels and only (12%) of study population they reach the university level **(Table2).**

**Table (2): The distribution of study population according to the educational level**

| Educational level | No | Frequency |
|---|---|---|
| Illiterate | 2 | 4% |
| Elementary | 22 | 44% |
| Secondary | 20 | 40% |
| University | 6 | 12% |
| Total | 50 | 100% |

The percentage of hand carriage of *Staphylococcus aureus* among study population was (38%) **(Table3).**

**Table (3): The percentage of hand carriage of *Staphylococcus aureus* among study population.**

| Food handlers | Hand carriage | |
|---|---|---|
| | No | Percentage |
| ***S. aureus* carriage** | 19 | 38% |
| No *S. aureus* carriage | 31 | 62% |
| Total | 50 | 100 |

In this study (58%) of the study population were carrying *Staphylococcus aureus* in their nostrils (**Table4)**.

**Table (4): The percentage of nasal carriage of *Staphylococcus aureus* among study population.**

| Food handlers | Nasal carriage | |
|---|---|---|
| | No | Percentage |
| *S. aureus* carriage | 29 | 58% |
| No *S. aureus* carriage | 21 | 42% |
| Total | 50 | 100 |

There was no statistically significant association between carrying of *Staphylococcus*aureus on hand and education level (*Pvalue*=0.2) **(Table5).**

**Table (5): The percentage of hand carriage of *Staphylococcus aureus* among different educational level of food handlers.**

| Educational level | Hand carriage | | *p value* |
|---|---|---|---|
| | No | Percentage | |
| Illiterate ( n=2) | **0** | **0** | **0.2** |
| Elementary (n=22) | **7** | **31%** | |
| Secondary (n=20) | **9** | **45%** | |
| University (n=6) | **3** | **50%** | |

The study denoted that there is no statistically significant association between carrying of *Staphylococcus aureus* on nose and education level (*Pvalue*=0.5) **(Table6).**

**Table (6). The percentage of Nasal carriage of *Staphylococcus aureus* among different educational levels of food handlers.**

| Education level | Nasal carriage | | *p value* |
|---|---|---|---|
| | No | Percentage | |
| Illiterate (n=2) | 1 | 50% | **0.5** |
| Elementary (n=22) | 12 | 54% | |
| Secondary (n=20) | 14 | 70% | |
| University (n=6) | 2 | 33% | |

There was statistically significant association between carrying of *Staphylococcus aureus* on hand and service year (*P value*=0.01) **(Table7).**

**Table (7): The percentage of Hand carriage of *Staphylococcus aureus* among food handlers according to service years.**

| Service years | Hand carriage | | p value |
|---|---|---|---|
| | No | Percentage | |
| <5 (n=39) | 11 | 28% | 0.01 |
| 5-10 (n=8) | 0 | 0% | |
| >10 (n=12) | 8 | 66% | |

The study found that there is no statistically significant association between service year and carrying of bacteria on nose (*P value*=0.12) **(Table 8).**

**Table (8): The percentage of nasal carriage *of Staphylococcus aureus* among food handlers according to service years.**

| Service year | Nasal carriage | | p value |
|---|---|---|---|
| | No | Percentage | |
| <5 (n=39) | 17 | 43.6% | 0.124 |
| 5—10 (n=8) | 7 | 87.5% | |
| >10 (n=12) | 5 | 41% | |

This study denoted that there was statistically significant association between carrying of *Staphylococcus aureus* on hand and job position (*Pvalue*=0.99) **(Table 9).**

**Table (9): The percentage of hand carriage of *Staphylococcus aureus* among food handlers according to job position.**

| Job position | Hand carriage | | p value |
|---|---|---|---|
| | No | Percentage | |
| Cooker (n=37) | 14 | 37.8% | **0.99** |
| Waiter (n=8) | 3 | 37.5% | |
| Cleaner (n=5) | 2 | 40% | |

The study found thatthere is no statistically significant association between carrying of *Staphylococcus aureus* on nose and job position (*P value*=0.09) **(Table 10)**

**Table (10): The percentage of nose carriage of *Staphylococcus aureus* among food handlers according to job position.**

| Job position | Nasal carriage | | p value |
|---|---|---|---|
| | No | Percentage | |
| Cooker (n=37) | 20 | 54% | 0.09 |
| Waiter (n=8) | 5 | 62% | |
| Cleaner (n=5) | 4 | 80% | |

**Discussion**

In this study out of 50food handlers work in different cafeterias of Atbara market, who were examined 29(58%) were found to be harboring *S. aureus* in their nostrils, this result was slitly similar to result reported in Botswana by (Loeto*et al.,* 2007),who found that the result of nasal carriage of *Staphylococcus aureus* was( 44.6%).[10] However, this finding was found to be higher than that reported in Sudan by (Saeed and Hamid, 2010), who denoted that the rate of

*Staphylococcus aureus* carriage was 21.6%. This variation has been attributed to the differences in the environmental and personnel hygiene of the study population.[8]

In this study out of 50 food handlers the hands of 19(38%) were contaminated with *S. aureus*, this finding similar to that reported in Egypt by (Bassyouni *et al.,* 2012)as they found *S. aureus* on the hands of (36.1%) of food handlers work at fayoum university.[11]

However this result is lower than that reported in South Africa by (Lues and Tonder, 2007) as they found 88% of food handlers hands were contaminated with *S. aureus* this variation in studies has been attributed to the differences in the degree of safety measure and hand washing habits in different geographic regions.[6]

Bacterial hand contamination rate in this study, showed significant association with service years(P value=0.013), in which the highest rate of hand carriage was found among those who worked form more than 10 years. This finding was in difference with study done in Ethiopia by (Asseefa *et al.,* 2015), who found that the highest carriage rate was among those who serve for less than 2 years.[2]

In this study *Staphylococcus aureus* nasal carriage rate was insignificantly associated with service years (*Pvalue*=0.124), this This result was in agreement with study done in Ethiopia by (Getnet*et al*., 2016) as they found that there were no significant association between service year and carriage of *Staphylococcus aureus* on nostril.[12]

In this study there was no significant association between job position and carriage *of Staphylococcus aureus* on hand (P value=0.995%). The rate of *Staphylococcus aureus* hand carriage was higher in cleaners (40%) than cookers (37.8%) and waiters (37.5%), this result was similar to finding obtained by (Asseefa *et al*., 2015) in Ethiopia, who found that the rate was higher also in cleaner (58%) than cookers (43.8%),and waiters (48.5%).[2]

In this study there was no significant association between job position and carriage of *Staphylococcus aureus* on nostril (P=0.094%) This result was in disagreement with study done in Ethiopia by (Getnet*et al,*(2019) as they found that there was significant association between job position and carriage of *Staphylococcusaureus* on nose(P value0.0013).This difference may be due to difference in Geographical area and the level of aware ness among the food handlers. [12]

In this study there was no association between education level and carriage of *Staphylococcus aureus* on hand (p=0.500).This result was in disagreement with study done in Ethiopia by (Asseefa*et al.,* 2015). As they found significant association between educational level and contamination of on hand, in which the rate of carriage was highest among illiterate persons 14(50%).[2]

In this study there was no association between education level and carrying of *Staphylococcus aureus* on nostril (p=0.225). This result is in agreement with study done in Ethiopia by (Getnet*et al*., 2019) as they found that there was no association between service year and carriage of *Staphlococcusaureus* on nose and education level.[12]

**Conclusion**

The study concluded that:Carriage of *Staphylococcusaureus* on hand was (38%) and in nose was (58%) ,also found that there was no significant statistical association between educational level and carriage of staphylococcus on hand, and nose,there was no significant statistical association between service year and carriage of staphylococcus on nose, but there was significant statistical association between service year and carriage of *Staphylococcus* on hand, and there is no statically significant association between service year and carriage of *Staphylococcus* on nose, and there was no significant statistical association between job position and carriage of *Staphylococcusaureus* on hand, and nose.

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Research Article

# *Serodetection of Herpes Simplex Virus Type 2 Infection among Pregnant Women in River Nile State*

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## Abstract

**Introduction**: Genital herpes remains a major problem causing considerable abortion among pregnant women and a higher risk to their infants leading to serious complication including intrauterine growth retardation, intrauterine fetal death, preterm labor, spontaneous abortion, congenital and neonatal herpes infections. The objective of this study to determine the prevalence of HSV-2 virus among pregnant women in River Nile State in Sudan.Method: A total of 75 pregnant women their age ranged from 16 to 44 years included in this study. 3 ml of whole blood was collected from each pregnant woman in heparin, Plasma was obtained by centrifugation, the plasma examined for the presence of Herpes simplex virus type-2 by immune Chromatography Test. **Results**: The result showed that out of 75 blood specimens investigated, 28(37.3%) were positive for HSV-2 infection, 25(89.3%) result positive for IgG antibody of Herpes simplex virus 2 and only 2 ( 7.1%) positive for IgM and 1 (3.6%) positive for both IgG and IgM. Conclusion: The study concluded that there is no statistically significant association between HSV-2 infection with age, trimester of gestation, abortion and educational level.

**Keywords:** ***Serodetection, HSV2, Pregnant Women ,River Nile State.***

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## Introduction

*Herpes simplex virus (HSV)* infection is one of most common viral sexually transmitted disease (STD) worldwide. Most genital herpes is caused by *HSV-2* and is almost transmitted sexually . [1]

*HSV-2* is the leading cause of genital ulcer disease in the developing countries. *HSV-2* infection is highly prevalent worldwide although most infected persons are either a symptomatic or have genital symptoms that remain unrecognized . [2]

Herpes viruses derived name from the Greek word herein, which means to creep. They are double strand DNA virus that belongs to the family *Herpesviridae*, subfamily Alphaherpesvirinae. Herpes virus cause persistent infection in human around the world . [3]

*HSV-2* infection in pregnant women can lead to abortion, premature labor, congenital and neonatal herpes (Egbagba, 2015). Worldwide the number of individuals seropositive for HSV-2 increase with age (27-28) (Whitley, 2006). *HSV-2* seroprevalence in the US has increased by 30%, comparing the developing countries, substantially higher rates of *HSV-2* has been observed in sub-Saharan Africa, where prevalence in adult range from 30% to 80% in women. Prevalence in the general population of Asian countries shows lower values, from 10% to 30% . [4]

Congenital HSV infection is rare but is higher than that of congenital syphilis, toxoplasmosis, and congenital rubella in years in which the virus was not epidemic. It shares clinical features such as microcehaly, hydrocephalus, and chorioretinitis with other congenital infections and is usually manifested by clinical abnormalities at birth. Postnatal acquisition of *HSV* is almost always due to *HSV-1* and is associated with contact with hospital personnel or family members who are shedding *HSV-1* Most neonatal infections result from exposure to HSV during delivery although in uteri and postnatal infections occasionally occur . [5]

Most cases of genital HSV infection in women occur without signs or symptoms of disease and are associated with cervical viral shedding . [5] The aim of this study was to determine the frequency of Herpes Simplex Virus type 2 (HSV-2) infection among pregnant women in River Nile State.

**Method:**

**Specimen collection:**

Following aseptic applications , 3 ml of venous blood were obtained from each subject , and plasma were extracted by centrifugation at 3000 rpm .

**TORCH test :**

The pouch was brought to the room temperature before opening it then the test cassette was removed from the sealed pouch and used it as soon as possible. the test cassette was placed on a clean level service .1 full drop of plasma was transferred and 2 drops of buffer to each specimen well of the test cassette respectively and then the timer was started. Trapping air bubbles in the specimen was Avoiding. Then the colored line(s) appear. The results should be read at 15 minutes and should not interpret the results after 20 minutes .

**Result interpretations**

**Negative test:** Red or pink line only at control region.

**Positive test:** Red or pink lines in both regions (at control and test).

**Invalid test:** no red or pink line at control region.

**RESULTS**

In this study a total of 75 pregnant ladies were enrolled for the study, their age ranged from 16 to 46 years, about (57.3%) of them belong to age group (25 to 46) years. Most of study participants were from rural areas 73.3% and only 26.7% were from urban areas. In this study 10.7% of study participants were illiterate, 33.3% reached primary school level, 24% at secondary school level and about 32% already completed their university study. In this study the frequency of *HSV2* among pregnant ladies was 37.3% **(Table 1).**

**Table ( 1): The frequency of HSV2 among the pregnant ladies:**

| HSV2 | Frequency | Percent |
|---|---|---|
| Positive | 28 | 37.3% |
| Negative | 47 | 62.7% |
| **Total** | **75** | **100%** |

Out of all positive cases only 2 (7.1%) were positive for IgM, and 25(89.3%) were positive for IgG and 1( 3.6%) were positive for both IgM and IgG **(Table 2).**

**Table (2): Distribution of *HSV2* antibodies among positive cases**

| HSV2 | Frequency | Percent |
|---|---|---|
| IgM | 2 | 7.1% |
| IgG | 25 | 89.3% |
| IgM + IgG | 1 | 3.6% |
| Total | 28 | 100% |

The study showed that there was no significant statistical correlation between HSV2 infection and age (p. value = 0.648) as in **(Table 3),** trimester (p. value = 0.308) **(Table 4)**, area of residence (p. value = 0.428) **(Table 5)**, abortion ( p. value= 0.141) **(Table 6)** and educational level ( p .value 0.758) **(Table 7)** .

**Table (3): Correlation of *Herpes Simples Virus 2* with age group**

| Age group | Frequency | Percent % | P .value |
|---|---|---|---|
| 16 -25 years(n=32) | 11 | 34.4% | 0.648 |
| 26 – 46 years(n=43) | 17 | 39.5% | |

**Table (4): Correlation of *Herpes Simples Virus 2* with gestational trimester**

| Trimester | Frequency | Percent % | P .value |
|---|---|---|---|
| First (n=17) | ٩ | 52.9% | ٠,٣٢٧ |
| Second (n=35) | ١١ | 31.4% | |
| Third (n=23) | ٨ | 34.8% | |

**Table (5): Correlation of *Herpes Simples Virus 2* with abortion**

| Abortion | Frequency | Percent % | P .value |
|---|---|---|---|
| Yes (n=32) | 15 | 46.9% | ٠,١٤١ |
| No (n=43) | 13 | 30.2% | |

**Table (6): Correlation of *Herpes Simples Virus 2* with abortion**

| Educational level | Frequency | Percent % | P .value |
|---|---|---|---|
| Illiterate(n=8) | 3 | 37.5% | |
| Primary(n=25) | 11 | 44% | |

| | | | |
|---|---|---|---|
| Secondary(n=18) | 5 | 27.8% | .758 |
| University and above(n=24) | 9 | 37.5% | |

**Discussion:**

In this study the frequency of *HSV-2* infection among pregnant women was 37.3%, this result was higher than the result obtained by (Basim, 2013) in Baghdad who reported that prevalence of *HSV-2* among pregnant women was 4.76% and this difference may be due to difference in study area.[6]

This study revealed that *HSV-2* among pregnant women was 2 (7.1%) for IgM, this result is lower than result obtained by (Maysaa and Hossam, 2007) in Egypt, who revealed that (40%) of study participants were IgM positive for *HSV-2*. This difference may be attributed to different techniques used for detection of the virus TORCH and ELISA techniques. [7]

A high frequency of *HSV-2* IgG (89.3%) was detected in pregnant women. This finding was similar to finding of study conducted among pregnant women at Omdurman Maternity Hospital by (Elsiddig, 2015) where the prevalence of *HSV-2* IgG was (91.1%). [8]

In this study there was no significant statistical correlation of HSV-2 infection with Age (p. value = 0.64) and this was similar to study conducted among European pregnant ladies by (Ferenc, 2011) who revealed that there is no significant statistical correlation between *HSV2* infection with age ( p. value =0.12). [9]

In this study there was no significant statistical correlation of *HSV-2* infection with trimester of gestation( p. value = 0.648), this is similar to study conducted by (Aminu, 2014) in Nigeria who concluded that there was no significant statistical correlation of *HSV-2* infection with gestational trimester (p. value=0.260). [10]

In this study there was no significant statistical correlation of *HSV-2* infection with history of abortion (p. value =0.141) and this is differ from result obtained in Iraq by (Musa, 2018) who reported that there was significant statistical correlation of *HSV-2* infection with history of abortion (p. value =0.03) this difference may be due to the difference in sample size between the two studies. [11]

The current study found that there was no significant statistical correlation of *HSV-2* infection with educational level (P. value=0.758) this finding was similar to result obtained by (Ahmed *et al* 2021) in Sudan (P. value=0.43), the similarity may be due to the same study area. [12]

## Conclusion

The frequency of *HSV-2* among pregnant women in River Nile State was 37.3%, and there were no significant statistical correlation of *HSV-2* infection with age, gestation trimester, previous abortion and educational levels.

## Ethical considerations

Informed consent was obtained from the enrolled pregnant ladies and the study objectives and the method of sample collection were explained, the confidentiality of the laboratory results were guaranteed for all participants

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Research Article

# *Serodetection of Helicobacter pylori* infection among gastritis patients in Atbara city

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**Abstract:**

**Introduction:** This was descriptive cross sectional hospital based study, done in Atbara city from July to October 2021; the aim of this study was to determine the percentage of *H .pylori* infections among gastritis patients in Atbra city. **Method:** A total of 100 stool specimens were collected from patients with gastritis in clean dry containers, the stool specimen was prepared by mixing approximately 50 mg of feces with the extraction buffer, then 2 drops were transferred to the specimen well on ICT cassette and the result was read within 10 minutes. **Results:** The study revealed that 89% of cases were *H .pylori* positive, while only 11% were negative for *H .Pylori.* The study also showed that there was significant statistical correlation between *H. pylori* infection and gender among gastritis cases (p value= 0.01), in which the frequency of *H. pylori* infection was higher in females (57%) than males (32%). The study also denoted that there were no significant statistical correlation between *H. pylori* infection and educational level, the source of food, family history of gastritis, family history of cancer and patient history for peptic ulcer. **Conclusion:** The study concluded that the frequency of *H .pylori infection among* gastritis patients *was* high. There was significant statistical correlation between *H .pylori* infection and gender among gastritis patients.

**Keywords: Serodetection, *Helicobacter pylori,* gastritis, Atbara city.**

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## Introduction:

Gastritis is aggravation, disturbance, or disintegration of the covering of the stomach. It can happen abruptly (intense) ,or slowly (ongoing). [1] Helicobacter pylori (H. pylori) are sort of microorganisms that causes disease in stomach. It is the primary driver of peptic ulcers, and it can cause gastritis and stomach malignant growth. H. Pylori cause persistent irritation by attacking covering of the stomach and creating cytotoxin named vacuolating cytotoxin (Vac-A) and subsequently can prompt ulcer arrangement. [2]

CagA is very much perceived oncoprotein which is infused into have cells by means of a pilus structure called type IV discharge framework, The quality locus that encodes CagA called cag pathogenicity island , After being infused into have cells, CagA adjusts intracellular sign transduction pathways that works with dangerous change of gastric epithelial cells. [3]

H. pylori infection is obtained during youth and continues deep rooted without a trace of treatment with anti-toxins. The greater part of the contaminated people stay asymptomatic for a significant stretch. Thus, long haul colonization of H. pylori can harm the gastric mucosa causing different illnesses of the upper gastrointestinal lot like ongoing gastritis, peptic ulcer, and gastric malignancies, especially gastric disease and gastric mucosa-related lymphoid tissue lymphoma. H. pylori has been perceived as a Class I cancer-causing agent by the International Agency for Research on Cancer and as one of the most grounded realized risk factors for gastric malignancies. [4]

Around 89% of all gastric tumors credited to H. pylori infection and annihilation of this contamination has known to lessen gastric malignant growth occurrence. Gastric malignant growth is positioned 6th in frequency and second in mortality among all tumors overall in view of tmost ongoing worldwide disease insights in 2018. [4]

H. pylori prevalence ranges somewhere in the range of 85% and 95% in emerging nations and somewhere in the range of 30 and half in created nations. The study of disease transmission of H. pylori infection has changed with upgrades in disinfection and strategies for annihilation. Nonetheless, the predominance is still plentiful. In non-industrial nations, the pervasiveness stays the most elevated, which is connected with financial status and levels of cleanliness. [4]After the year 2000, the pervasiveness

of H. pylori became lower than before in European nations. In any case, in Asia, the predominance continues as before. To this date, 4.4 billion people around the world are assessed to be H. pylori-tainted. The region with the most elevated detailed predominance was accounted for in Africa (70.1%) while the least commonness was accounted for in Switzerland (18.9%). In Southern Asia, Pakistan and India have shown the highest H. pylori prevalence (81% and 63.5%, individually). In Western Asia, Turkey introduced the most elevated commonness (77.2%). [4]

**Method:**

**Specimen collection:**

The patient was given clean container for collection of stool sample, to bring small amount of fecal specimen without contamination with urine specimen, and not to contaminate the outer surface of container.

**Procedure:**

The test cassette, buffer left to reach room temperature prior testing, the cap of specimen collection tube were un screw, fecal specimen were stabbed by collection applicator to collect approximately 50mg of feces, the specimen collection tube shake to mix the extraction buffer with the specimen. The tube is left for 2 minutes and the test cassette were removed from foil, and transfer 2 drops to specimen well, the result read at 10 minutes.

**Reading and Interpretation of the Results**

**Positive result:** if both C band and T band are developed the test is indicate for the presence of *H .Pylori* antigen in the specimen. The result is positive.

**Negative result:** if only the C band is developed, the test indicates that no detectable *H .Pylori* antigen is present in the specimen. The result is negative.

**Invalid:** if no C line is developed, the assay is invalid regardless of any color development on the T line.

**False –negative results:** may be obtained on specimens from patients who have ingested selected medications (anti-microbial, proton pump inhibitors, and bismuth preparations) within the 2 weeks prior to specimen collection. If clinically indicated, the test may be repeated on a new specimen obtained 2 week after stopping treatment with these medications. [5]

**Results:**

A total of 100 stool samples were collected from gastritis patients in Kamal altoum compound, life care compound and Atbara

teaching hospital. In this study most of study populations were females (60%) compared to males (40%). The age of study participants were ranged from 15-30 years (19%), 31-45 (24%), and more than 75 years old, most of them were in age group more than 45 years (57%).

The study revealed that the frequency of *H .pylori* infection among gastritis patients was (89%). (**Table.1**)

**Table.1: The frequency of *H. pylori* infections among gastritis patients**

| ***H .pylori* infection** | **No** | **Frequency** |
|---|---|---|
| Positive | 89 | 89% |
| Negative | 11 | 11% |
| Total | 100 | 100% |

In this study there was significant statistical correlation of *H. pylori* infection with participant gender (p-value 0.01), in which the percentage of infection was higher in females (57%) than males (32%). (**Table 2**)

**Table (2): The correlation between *H. pylori* infection and gender among gastritis patients**

| **Gender** | ***H. pylori* infection** | | ***p value*** |
|---|---|---|---|
| | **Positive** | **Negative** | |
| Male | 32(32%) | 8(8%) | |
| Female | 57(57%) | 3(3%) | **0.019** |

The study revealed that there was no significant statistical correlation of *H. pylori* infection with age (P value=0.1) **Table (3)**, eating source (p value=0.7) **Table (4),** history of peptic ulcer (p value=0.2) **Table (5),** history of gastritis (p value=0.06) **Table (6)**, or history of cancer (p value=0.2). **Table (7)**

**Table (3): The correlation between *H. pylori* infection and age among gastritis patients.**

| **Age** | ***H .pylori* infection** | | ***p value*** |
|---|---|---|---|
| | **Positive** | **Negative** | |
| >30 | 18(18%) | 1(1%) | |
| 30-45 | 19(19%) | 5(5%) | 0.1 |
| < 45 | 52(52%) | 5(5%) | |

**Table (4) : The correlation between *H .pylori* infection and eating source among gastritis patients**

| Eating source | *H .pylori* infection | | *p value* |
|---|---|---|---|
| | Positive | Negative | |
| Home | 53(53%) | 6(6%) | **0.7** |
| Restaurant | 36(36%) | 5(5%) | |

**Table (5): The correlation between *H .pylori* infection and those who previously have ulcer among gastritis patients**

| History of gastric ulcer | *H .pylori* infection | | p value |
|---|---|---|---|
| | Positive | Negative | |
| Yes | 48(48%) | 8(8%) | **0.2** |
| No | 41(41%) | 3(3%) | |

**Table (6): The correlation between H *.pylori* infection and family history of gastritis among gastritis patient**

| Family history of gastritis | *H .pylori* infection | | p value |
|---|---|---|---|
| | Positive | Negative | |
| Yes | 56(56%) | 10(10%) | **0.06** |
| No | 33(33%) | 1(1%) | |

**Table (7): The correlation between *H .pylori* infection and family history of cancer among gastritis patients**

| Family history of cancer | *H .pylori* infection | | p value |
|---|---|---|---|
| | Positive | Negative | |
| Yes | 9(9%) | 0(0.0%) | **0.2** |
| No | 80(80%) | 11(11%) | |

**Discussion:**

In this study out of 100 gastritis patients, (89%) were *H .pylori* positive and only (11%) were *H .pylori* negative, this finding was differ from result obtained by Palmer *et al* in North West province of Cameron, who showed a lower frequency of *H. pylori* infection (72%). [6]

The study denoted that there was significant correlation between gender and *H .pylori* infection (p-value 0.01), these result was similar to that of (Tay *et al*, 2008) who denoted that there was significant correlation of *H .pylori* infection (p-value 0.02). However other study conducted by (Nathan *et al.,* 2018), found that there was no significant correlation of infection with gender. This difference may be attributed to very big sample size applied by (Nathan *et al.,* 2018) which was 500 participants in comparison to only 100 participants in this study. [7], [8]

In this study the highest rate of infection was noticed among age group more than 45 years old (52%), and declared that there was no significant correlation between *H .pylori* infection and age (p-value 0.1). This result was similar to result obtained by (Adlekha *et al.,* 2013) in Karla, India, who indicate that there was no significant statistical difference in age related to *H. pylori* with (*P* -value 0.93). [9]

The frequency of *H. pylori* infection among illiterate participants was (28%), in elementary level was (21%), in secondary level was (16%) and in university level was (24%), the results showed that there were no correlation of *H .pylori* infection with educational level of participants (p-value 0.4). Similar results were obtained by (Rahul *et al*., 2013,) who found that education is not associated with disease status (*P* value = 0.23). This may be attributed to absent of commitment with personal hygiene rules

including hand washing measures among both educated and non-educated persons. [10]

Results of this study showed that the occurrence of *H .Pylori* infection among participants had no statistical significance with places of taking meals (p-value 0.7). This finding is same to study done by (Kathleen *et al.,*; 2002) in Bolivia who found that the place of food consumption was not significant in the transmission of *H .pylori*. This fact may indicate that *H. pylori* transmission is mostly depend on personal hygiene behaviors especially hand washing behavior rather than other sources of contamination. [11]

The study revealed that there was no significant statistical correlation between patient history of gastric ulcer and *H. pylori* infection. This result is totally differed from that revealed by (Jose *et al*., 2007) in university hospital in South east Brazil, who showed that there is significant correlation between *H .pylori* infection and those who have peptic ulcer before (p-value 0.001). [12]

The frequency of *H. pylori* infection among participants with family history of gastritis infection was higher (56%) than those with no family history of gastritis (33%)**,**The study showed that there was no significant correlation between *H. pylori* infection and family history of gastritis (p-value 0.06). This result was same to result revealed by (Alebia and Kaba, 2016) , who found that there is no statistical significance correlation between family history of gastritis and *H .pylori* infection(p-value 0.9). [5]

Results also showed that family history of cancer shows no correlation with *H .pylori* infection (p-value 0.2). This finding is same to result of study done by (Tovey *et al.,* 2004) in India that reported no association between stomach cancer and *H. pylori* infection (p-value 0.4) . [13]

**Conclusion:**

The study concluded that the frequency of *H .pylori infection among* gastritis patients *was* high. There was significant statistical correlation between *H .pylori* infection and gender among gastritis patients.

The frequency of *H. pylori* infection was highest among age group of more than 45 years. Frequency of *H. pylori* among illiterate was nearly equal to that of university level among gastritis patients. There were no significant statistical correlations between *H. pylori* infection with family history of gastritis or gastric carcinoma.

**Ethical Consideration:**

All participants were informed about objectives and purposes of the study, also informed about sample collection procedure, possible discomfort and freedom to choose either to participate or decline to take part in the study.

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# إستخدام النماذج غير الخطية للتنبؤ بالطاقة الكهرومائية المولدة بالشركة السودانية للتوليد المائي

# Using Non-linear Models to Predict the Hydro Power Generation in SHGC

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**المستخلص:**

تم في هذه الدراسة إستخدام النماذج الغيرالخطية لدراسة وتحليل الطاقة الكهرومائية المنتجة بالشركة السودانية للتوليد المائي للفترة من (١٩٦٥-٢٠٢١) لما تمتاز به هذه النماذج من دقة ومرونة عاليتين في تحليل الظواهر. حيث تم استخدام المنهج الوصفي والتحليلي للوصول الي نتائج الدراسة باستخدام برنامج (SPSS- 24) و Minitab 17، أظهرت نتائج التطبيق ان النموذج الملائم والكفؤ هو نموذج القوي و وفقاً لنتائج تقدير هذا النموذج تم التنبؤ بالطاقة المولدة بالشركة السودانية للتوليد المائي للفترة من ٢٠٢٢-٢٠٣٣ ، حيث أظهرت هذه القيم تناسقاً مع مثيلاتها في القيم الأصلية.

***الكلمات المفتاحية: الطاقة، التوليد المائي، النماذج غير الخطية***

**Abstract**

In this study, we used nonlinear models to study and analyze the hydro power Generation produced by the Sudanese hydro power generation Company for the period (1965-2021), these models have high accuracy and flexibility to analysis the phenomena. Where the descriptive and analytical approach was used to reach the results of the study using the program (SPSS-24) and Minitab 17, the results of the application showed that the appropriate and efficient model is a Power model and according to the results of the estimate of this model was predicted the energy generated by the Sudanese hydro power Generation Company for the period from 2022-2033, where these values showed consistency with their original values.

**Keywords:** *Hydro-Power, Power, Power Model, Non-Linear Model*

## المقدمــة:

تعتبر النماذج الغير خطية من الاساليب الإحصائية المهمة في تحليل الكثير من الظواهر، وتتلخص أهم أهدافها في الحصول على وصف دقيق للظاهرة وبناء نموذج مناسب لتفسير سلوكه وإستخدام النتائج للتنبؤ بسلوك الظاهرة في المستقبل.

وإيماناً منا بالدور المتعاظم الذي تلعبه الطاقة الكهربائية كونها المصدر الرئيسي للتنمية المستدامة، ارتأينا القيام بإعداد دراسات وتطبيقات إحصائية على الطاقة الكهربائية المنتجة بواسطة المياه بإستخدام النماذج الغير خطية، ليمكن الجهات المختصة من تحليل الظاهرة وتحديد إتجاهاتها المستقبلية و وضع الخطط اللازمه لها.

## أهمية البحث :

تتجلي أهمية البحث من خلال إتباع إسلوب علمي متقدم لبناء نموذج إحصائي يمكن الجهات القائمة على التوليد المائي والجهات التخطيطية الأخرى من معرفة نمط التوليد الآن وما ستؤول إليه في المستقبل .

## فروض البحث:

هنالك استجابة غير خطيه في إنتاج الطاقة الكهرومائية مع مرور الزمن .

## أهداف البحث:

يهدف البحث إلى إيجاد نموذج إحصائي يوضح الطاقة المولدة بإستخدام النماذج الغير خطية القابلة للتحويل.

## الحدود الزمانية المكانية:

تتمثل الحدود الزمانية في الفترة من (١٩٦٥-٢٠٢١) .

أما الحدود المكانية فتتمثل في محطات التوليد المائية (الروصيرص ، سنار، خشم القربة، جبل اولياء) .

## منهجية البحث والأدوات المستعملة:

تستخدم الدراسة منهج التحليل الوصفي في وصف متغيرات الدراسة بالإضافة للتحليل الإستنتاجي المسئول عن تحقق فرضيات الدراسة من عدمة بإستخدام الأدوات الإحصائية الخاصة بالنماذج الغير خطية معتمداً على النظرية الإحصائية وذلك بإستخدام الحزم الإحصائية الجاهزه Microsoft Excel, SPSS Ver.24, Minitab 17.

## ١. الجانب النظري :

للتنبؤ الدقيق أهمية بالغة ولذلك فقد أهتم الباحثون وذو العلاقة بالدراسات والبحوث التنبؤية و وضعوا العديد من الطرق والنماذج التنبؤية كان ابرزها النماذج الغير خطية التي

أثبتت كفائتها ودقتها في مجالات تطبيقها، ولذلك سنتناول دراسة النماذج الغير خطية ومعايير المفاضلة بينها.

**١-١ النموذج الخطي**

$$y_i = b_o + b_1 x_i + e_i \dots\dots (1)$$

**١-٢ النماذج غير الخطية :**

**١- النموذج التربيعي Quadratic Model**

ومعادلته كالآتي:

$$y_i = b_o + b_1 t_i + b_2 t_i^2 + e_i \dots\dots (2)$$

ومن الملاحظ أن النموذج أعلاه غير خطي وبعد تحويله إلى خطي تكون المعادلة كالآتي:

$$y_i = b_o + b_1 t_i + b_2 x_i + e_i \dots\dots (3)$$

حييث $x_i = t_i^2$

**٢- النموذج التكعيبي : Cubic Model**

$$y_i = b_o + b_1 t_i + b_2 t_i^2 + b_3 t_i^3 + e_i \dots\dots (4)$$

ومعادلته المحولة إلى خطية تكون كالآتي :

$$y_i = b_o + b_1 t_i + b_2 x_{1i} + b_3 x_{2i} + e_i \dots\dots (5)$$

حيث $x_{1i} = t_i^2$ و $x_{2i} = t_i^3$

**٣- نموذج القوي : Power Model**

$$y_i = b_o + t_i^{b1} + e_i \dots\dots (6)$$

ومعادلته المحولة إلى خطية تكون كالآتي :

$$y_i^* = b_o^* + b_1 t_i^* + e_i \dots\dots (7)$$

**حيث** $y_i^* = Lny_i$ و $Lnb_o = b_o^*$ و $Lnt_i = t_i^*$

**٤- النموذج الاسي: Exponential Model**

$$y_i = b_o + e^{b1 t_i} + e_i \dots\dots (8)$$

ومعادلته المحولة إلى خطية تكون كالآتي :

$$y_i^* = b_o^* + b_1 t_i + e_i \dots\dots (9)$$

**حيث** $y_i^* = Lny_i$ و $Lnb_o = b_o^*$

**٥- النموذج اللوغاريثمي : Logarithmic Model**

**ومعادلتة تكون كالاتي :** $y_i = b_o +$ **.....(10)**

$b1\ Lnt_i + e_i$

**ومعادلتة المحولة الي خطية تكون كالاتي :** $y_i =$

$b_o + b1\ X_i + e_i \dots\dots (11)$

**حيث** $X_i = Lnt_i$

**٦- النموذج المعكوس: Inverse Model**

**ومعادلتة تكون كالاتى :** $Y_i$ $+e_i$ **.....(12)**

$= b_o + b_1 \frac{1}{ti}$

**ومعادلتة المحولة الى خطية تكون كالاتى :**

$Y_i = b_o + b_1 x_i + e_i$ **.....(13)**

**حيث :** $Xi = \frac{1}{ti}$

**٧- النموذج الشبيه بالحرف (S) : S-Shaped Model**

**ومعادلتة كالاتى :** $Y_i$ $+e_i$ **..... (14)**

$= e^{(bo+b1/ti)}$

**ومعادلتة المحولة الى خطية تكون كالاتى :**

$Y_i^* = b_o + b_1 x_i + e_i$ **.....(15)**

$Yi^* = \ln Yi$, $Xi = \frac{1}{ti}$ حيث :

**٨- النموذج النمو: Growth Model**

**ومعادلتة كالاتى:**

$Y_i = e^{(bo+b1ti)} + e_i$ .....(16)

**ومعادلته المحولة الى خطية تكون كالاتى: .....(17)**

$Y_i^* = b_o + b_1 t_i + e_i$

**حيث:** $Y_i^* = \ln Yi$

**٩- النموذج المركب : Compound Model**

**ومعادلته كالاتى:** $Y_i = b_o * b_1^{ti} + e_i$ .....(18)

**ومعادلته المحولة الى خطية تكون كالاتى: .....(19)** $Y_i^*$

$= b_o^* + b_1^* t_i + e_i$

**حيث:** $Y_i^* = \ln Yi$ , $b_o^* = \ln bo$ , $b_1^* = \ln b1$

**١٠- النموذج اللوجستى :Logistic Model**

**ومعادلتة تكون كالاتى:** $Y_i = \frac{1}{(\frac{1}{a}+bo*b1^{ti})} + e_i$ ....(20)

**ومعادلتة المحولة الى خطية تكون كالاتى :**

$Y_i^* = b_o^* + b_1 t_i + e_i$ .....(21)

**حيث:** $b_o^* = \ln bo$ , $b_1^* = \ln b1$ , $Y_i^* = \ln(\frac{1}{yi-\frac{1}{a}})$

**٣-١ تقدير النموذج: The Model Estimation**

بعد تحديد شكل النموذج لابد من تقدير معلمات النموذج و $\hat{b}_1, \hat{b}_0, \sigma^2$ وذلك بإستخدام البيانات التاريخية المتوفرة لدينا.

هنالك طرق كثيرة لتقدير المعلمات سنذكر منها طريقة المربعات الصغري.

**طريقة المربعات الصغرى:**

تعتبر هذه الطريقة من أكثر الطرق استعمالاً لتوفيق معادلة خط الاتجاه العام للبيانات المشاهدة وقد سبق الحديث عنها في باب الانحدار حيث x يمثل الزمن, y يمثل قيم الظاهرة موضع الدراسة. ومن مميزات هذه الطريقة ما يلي:

١ – تجعل مجموع الفروق بين قيم الظاهرة الفعلية والمقدرة من المعادلة مساوياً للصفر أي أن:

$$\sum_{i=1}^{n}(y_i - \hat{y}_i) = 0 \quad .....(22)$$

٢ – تجعل مجموع مربعات الفروق بين قيم الظاهرة الحقيقية وقيم الظاهرة المقدرة من المعادلة أقل ما يمكن أي أن مجموع مربعات الاخطاء تمثل نهاية صغرى, وفي حالة ما يكون الاتجاه العام على شكل خط مستقيم فإن معادلة هذا الخط تكون على الصورة:

حيث:

$$\hat{y}_i = a + bx_i \quad , \quad a = \bar{y} \quad .....(23)$$

$$b = \frac{\sum_{i=1}^{n} x_i y_i}{\sum_{i=1}^{n} x_i^2}$$

حيث: $x_i = t_i - \bar{t}$ ، t تمثل السنوات و $\bar{t}$ هي الوسط الحسابي للسنوات أو هي الوسط الحسابي لأول وآخر سنة.

**٤-١ معايير إختيار أفضل نموذج: The Best Model Select Criteria**

يتم مقارنة النماذج وفق معايير إختيار أفضل نموذج بغرض إيجاد أفضل نموذج ممثل لبيانات متغير الدراسة والمعايير المستخدمة للمفاضلة هي:

١- **اختبار اف: (F)**

ان الهدف الاساسى من إختبار F هو اختبار معنوية نموذج الانحدار او بمعنى اخر اختبار تاثيرالمتغير المستقل (فى النموذج الخطى البسيط ) هذا يعنى ان الفرضية سوف تكون كالاتى:

$H_o$: النموذج الاحصائي غير معنوي

$H_1$: النموذج الاحصائي معنوي

تتم مقارنة قيمة F المحسوبة مع قيمة F الجدولية المستخرجة عند درجة حرية للبسط تساوى واحد وللمقام (n-2) ومستوى معنوية $\propto$ فاذا كانت قيمة F المحسوبة اقل من قيمة F الجدولية ( $F_{1,n-2,\propto}$) فانه يتم قبول فرض العدم اى انه لا يوجد تاثير معنوى من قبل المتغير المستقل على المتغير المعتمد واذا كانت قيمة F المحسوبة اكبر من الجدولية فانه يتم رفض فرض العدم وقبول الفرض البديل اى انه لا يوجد تاثير معنوى من المتغير المستقل على التابع.

٢- **معامل التحديد: Coffined Of Determination**

يعرف معامل التحديد علي انه نسبه مساهمة المتغير المستقل (اوالمتغيرات المستقلة) في احداث التغيرات في المتغير المعتمد وهو يمثل مربع معامل الارتباط الخطي ويرمز لة بالرمز $R^2$ في شكل قانون $R^2=\frac{SSR}{SST}$ ومما سبق ذكره فان المساهمة في التغيرات في المتغير المعتمد تتم بسببين (الانحداروالخطأ) لذلك فان نسبه مساهمه الخطأ في احداث التغيرات في المتغيرالمعتمد يساوي ( $\frac{SSE}{SST}$ ).

وعلي سبيل المثال لوافترضنا ان المتغير المستقل هو الدخل والمتغير المعتمد هو الانفاق وكان $R^2=80\%$ هذا يعني ان نسبه مساهمه الدخل في احداث التغيرات علي الانفاق هي ٨٠٪ من الاختلافات في القيم للانفاق سببها الدخل وان ( ٢٠٪ ) من التغيرات في الانفاق هي بسبب المتغيرات العشوائية.

٣- **إحصائية مالو CP :**

يعتبر من أهم مقاييس المفاضلة بين النماذج الإحصائية لإختيار إفضل نموذج ممثل للبيانات ويكون النموذج صاحب أقل قيمة لإحصائية مالو هو النموذج الأفضل, وتحسب كالآتي :

$$C_p = \frac{SSE(X_1,X_2,\dots,X_P)}{MSE(X_1,X_2,\dots,X_m)} - (n-2p)$$

..... (24)

لاحظ أن p هو عدد المعلمات في النموذج ( ومن بينها $\beta_0$ أيضا) أي أن ($p = m+1$).

٤- **الخطأ القياسي: Stander Error**

هو ايضاً معيار يقيس الفرق بين القيم الحقيقية والمقدرة ويستخدم للمفاضلة بين النماذج بحيث يكون النموذج صاحب اقل خطأ قياسي هو النموذج الافضل في تمثيل البيانات موضع الدراسة ويحسب بالصيغة التالية:[٢]

$$SE = \sqrt{\frac{\sum_{i=1}^{n}(y-\hat{y})^2}{n-2}} \quad \text{..... (25)}$$

٥- **معامل عدم التساوي لـ ثايل: Theil's Inequality Coefficient**

لقياس دقة التنبؤات التي يتم الحصول عليها من النموذج القياسي المقدر, اقترح H.Theil المعيار الاتي:

$$= \frac{\sqrt{\sum_{t=1}^{n-1}\left(\frac{f_{t+1}-y_{t+1}}{y_t}\right)^2}}{\sqrt{\sum_{t=1}^{n-1}\left(\frac{y_{t+1}-y_t}{y_t}\right)^2}}$$

..... (26)

حيث البسط يمثل التغيير في القيم المتنبأ بها نسبة الي القيم الفعلية, والمقام يمثل التغيير النسبي في القيمة الفعلية.

واعتماداً على المعادلة (3-39) فإن قيمة U تاخذ المدى $\infty > U \geq 0$ , وكلما كانت قيمة U قريبة من الصفر فإن ذلك يعني اقتراب قيم البسط من قيم المقام , وهذا بدوره يعكس التقارب بين القيم التنبؤية $\hat{Y}_i$ والقيم الحقيقية $Y_i$, وفي هذه الحالة فإن النموذج القياسي يتمتع بقوة تنبؤية عالية, أي أن تنبؤاته تكون دقيقة جداً.

أما إذا كانت قيمة U كبيرة فهذا يعني وجود أختلاف كبير بين البسط و قيم المقام , وبالتالي تباعد القيم التنبؤية $\hat{Y}_i$ عن القيم الفعلية $Y_i$ , وفي هذه الحالة فإن النموذج القياسي سيعاني من ضعف القوة التنبؤية، أي أن تنبؤاته سيئة.

إذا كانت البسط = المقام فإن ذلك يؤدي الي U=0 , وفي هذه الحالة فإن التنبؤات تكون مضبوطة ولا تعاني من أية أخطاء, وتعتبر هذه الحالة مفصلة لدي الباحثين. أما إذا كانت البسط يساوي الصفر فإن ذلك يجعل U=1 وفي هذه الحالة فإن تنبؤات النموذج غير مقبولة بدرجة عالية من قبل الباحثين. وإذا حدث أن U>1 فهذا يعني أن القوة التنبؤية للنموذج سيئة جداً فهي أسوأ من الحالة السابقة, وهذه النتيجة تكون مرفوضة من قبل الباحثين, عموماً تقبل تنبؤات النموذج القياسي إذا كانت $1 > U \geq 0$ وكلما كانت U قريبة من الصفر كانت التنبؤات دقيقة.

**فحص وإختبار دقة النموذج: Model Diagnostics Checking**

بعد التعرف على نموذج مبدئي وتقدير معلمات هذا النموذج نجري بعض التشخيصات على البواقي أو أخطاء

التطبيق لنرى مدى مطابقة النموذج للسلسلة المشاهدة ، ويفترض أن البواقي هي مقدرات التشويش الأبيض $a_t$ والتي نفترض إنها موزعة طبيعياً بمتوسط صفري وتباين $\sigma^2$. البواقي تعطى بالعلاقة

$$e_t = z_t - \hat{z}_t = \hat{a}_t, \quad t = 1,2,...,n$$

يقوم الفحص والإختبارات على فحص البواقي هل هي تشويش أبيض أم لا ، فإذا كانت تشويش أبيض نعتبر النموذج المطبق مقبولاً أما إذا لم تكن كذلك فيجب علينا إعادة النظر وإقتراح نموذج آخر ولإجراء الفحص وإختبار الدقة, ويستخدم الإختبارات التالية لفحص الدقة.

١. **إختبار المتوسط للبواقي:**

$H_0: E(a_t) = 0$

$H_1: E(a_t) \neq 0$

وهو إختبار من طرفين ونستخدم الإحصائية U= $\frac{e}{se(e)}$ والتي لها توزيع طبيعي قياسي فعند مستوي معنوية α=0.05 نعتبر ان E(at) =0 إذا كانت I u I <1.96 (هذا علي إعتبار ان حجم العينة اكبر من ٣٠ وحدة وهذا دائماً متحقق للمتسلسلات الزمنية التي ندرسها)

٢. **إختبار عشوائية البواقي:**

نختبر عشوائية البواقي بواسطة إختبار الجري Runs test حول المتوسط وحول الصفر وهو احد الإختبارات اللامعلمية.

٣. **إختبار الترابط أو إستقلال البواقي:**

يختبر ترابط أو إستقلال البواقي بواسطة إختبار الترابط الذاتي Autocorrelation test وذلك بحساب ورسم الترابطات الذاتية للعينة SACF للبواقي ومقارنتها مع دالة الارتباط الذاتي التشويش الابيض.

H0: r1 =0 , H1: r1 ≠0

حيث الإحصائية $\frac{r1}{se(r1)}$ =U لها توزيع طبيعي قياسي فعند مستوي معنوية α=0.05 نعتبر ان r1 =0 إذا كانت I u I<1.96 .

٤. **إختبار طبيعة البواقي:**

نختبر في ما اذا كانت البواقي موزعة طبيعياً وذلك بعدة طرق مثل:

إختبار حسن التطابق Goodness of Fit Test ونستخدم الإختبار اللامعلمي كولموجوروف– سميرنوف Kolmogorov- Smirnov Test ، مخطط الإحتمال الطبيعي Normal probability Plot ، مخطط الربيعات– الربيعات Q-Q Plot .

٥. **إختبار الجودة (التطابق): ( $\chi^2$ ) Goodness Test**

هنالك شروط يجب توافرها في كل الإختبارات الإحصائية فمثلاً تطبيق إختبار t لمقارنة وسطي مجتمعين يعتمد على كون توزيع المجتمعات توزيع طبيعي والسؤال هنا كيف لنا ان نعرف ان نتحقق اذا كان هذا الشرط متحقق ام لا

وهنالك حالات أخري يراد فيها معرفة ماذا اذا كان المتغير يتبع توزيع معين مثل توزيع بواسون وذو الحدين وتتلخص فكرة إختبار جودة المطابقة في مقارنة عدد المشاهدات Oi والتي نراها من خلال التجربة مع عدد المشاهدات المتوقعة ei اذا كانت الفرضية المبدئية صحيحة، يتم حساب قيمة مربع كاي من هذا القانون

$$\chi^2 = \sum_{i=1}^{n} \frac{(o_i - e_i)^2}{e_i} \quad \dots (27)$$

ومن ثم يتم مقارنة بقيمة $\chi^2$ الجدولية بدرجة حرية df(n-) (1) وبعد ذلك يتم إتخاذ القرار المناسب برفض او قبول فرض العدم (Ho)

**٢. الجانب التطبيقي :**

في هذه الجزئية تم بناء نموذج لمتغير الدراسة (الطاقة الكهرومائية) بإستخدام النماذج الغير خطية وذلك بتقدير النماذج وحساب المقاييس المختلفة لها وإمكانية تقييمها ومن ثم إختيار أفضل نموذج ممثل للبيانات وإختبار معنوية المعلمات ومن ثم فحص النموذج من حيث الدقة لمعرفة مدي مطابقة القيم المقدرة للقيم الحقيقية واخيراً التنبؤ بما ستؤول اليه الظاهرة في المستقبل.

**٢-١ إختيار أفضل نموذج:**

**جدول رقم (١) يوضح النماذج الغير خطية البسيطة ومقاييس تقييمها**

| | F | SE | $R^2$ | Cp | U |
|---|---|---|---|---|---|
| LINEAR | 691.344 | 156.99 | 0.926 | 2 | 1.85 |
| LOGARITHMIC | 234.96 | 251.88 | 0.810 | 2 | 4.30 |
| INVERSE | 25.99 | 476.59 | 0.309 | 2 | 8.80 |
| QUADARATIC | 365.005 | 153.18 | 0.931 | 3 | 1.25 |
| CUBIC | 252.95 | 150.53 | 0.935 | 4 | 1.34 |
| COMPOUND | 164.975 | 0.478 | 0.75 | 2 | 2.42 |
| POWER | 841.822 | 0.236 | 0.938 | 2 | 1.14 |
| S | 74.044 | 0.623 | 0.574 | 2 | 4.636 |
| GROWTH | 164.975 | 0.478 | 0.75 | 2 | 2.42 |
| EXPONENTIAL | 164.975 | 0.478 | 0.75 | 2 | 2.42 |

**المصدر: إعداد الباحثان بواسطة برنامج SPSS & EXCEL ٢٠٢٢م**

ومن خلال الجدول رقم (3–2) نجد ان **نموذج القوي (Power)** هو النموذج الانسب لتمثيل البيانات وذلك لاحتواءه على اعلى نسبة معامل تحديد ($R^2$) واعلى قيمة (F) وإقل خطأ قياسي (SE) وإقل قيمة إحصائية مالو (Cp) وإقل معامل عدم التساوي ثايل (U).

### ٢–٢ إختبار معلمات ومعنوية النموذج:

**Model Summary**

| R | R Square | Adjusted R Square | Std. Error of the Estimate |
|---|---|---|---|
| .969 | .939 | .938 | .236 |

The independent variable is time.

**ANOVA**

| | Sum of Squares | df | Mean Square | F |
|---|---|---|---|---|
| Regression | 47.081 | 1 | 47.081 | 841.8 |
| Residual | 3.076 | 55 | .056 | |
| Total | 50.157 | 56 | | |

The independent variable is time.

**Coefficients**

| | Unstandardized Coefficients | | Standardized Coefficients | |
|---|---|---|---|---|
| | B | Std. Error | Beta | t |
| ln(time) | 1.021 | .035 | .969 | 29 |
| (Constant) | 32.634 | 3.699 | | 8 |

The dependent variable is ln(generation).

**المصدر: إعداد الباحث بواسطة برنامج SPSS ٢٠٢٢م**

اذا المعادلة المقدرة للنموذج اعلاه تصبح كالاتى :

$$Yt = 32.634*t^{1.021}$$

والمعادلة الخطية المقدرة لها هى:

$$Y_i^* = 3.433+1.021t^*$$

$$Y_i^* = Ln(Y_t) \quad ,T^*= Ln(t)$$

**إختبار معنوية المعلمات لنموذج القوي:**

ومن النتائج أعلاه نجد ان قيمة ( Sig =.0000) لإختبار (t) الخاصة بمعلمة الثابت (المقطع) أقل من قيمة مستوي المعنوية (٠.٠١) عليه فإن المقطع لنموذج القوي معنوي, وبما ان قيمة ( Sig =.0000) لإختبار (t) الخاصة بمعلمة الميل للمتغير المستقل (الزمن) أقل من قيمة مستوي المعنوية (٠.٠١) عليه فإن الميل للمتغير المستقل معنوي.

**إختبار معنوية نموذج القوي:**

من النتائج أعلاه نجد ان قيمة (Signif =.0000) لإختبار (F) الخاصة بمعنوية النموذج هى اقل من( 0.01) عليه فإن النموذج اعلاة معنوى عند مستوى معنوية ١٪ وصالح للتنبؤ.

وبما ان قيمة معامل التحديد (R2 =.93) هذا يعنى ان نسبة مساهمة عنصر الزمن في احداث التغير فى الطاقة المولدة من الماء تكون بنسبة ٩٣٪ ، بمعني أخر أن الزمن يساهم في إحداث التغيرات التي تحدث في التوليد المائي بنسبة ٩٣٪ وإن ٧٪ من المساهمة متغيرات أخري غير مضمنة في النموذج.

**٢-٣ فحص وإختبار دقة النموذج : Model Diagnostics Checking**

**١/ إختبار متوسط البواقي:**

**One-Sample Test**

Test Value

| | t | df | Sig. (2-tailed) | Mean Difference |
|---|---|---|---|---|
| Error for generation with time from CURVEFIT, MOD_2 POWER | .275 | 56 | .785 | 5.898 |

**المصدر: إعداد الباحث بواسطة برنامج SPSS ٢٠٢٢م**

بما ان القيمة الاحتمالية (Sig) تساوي (0.785) وهي قيمة اكبر من مستوي المعنوية (٠.٠٥) عليه نقبل فرض العدم إي ان المتوسط يساوي الصفر.

**٢/ الترابط الذاتي للبواقي:**

من الشكل رقم (٣) والشكل رقم (٤) نلاحظ ان نمط الترابط الذاتي والترابط الذاتي الجزئي يتبعان نمط خطأ التقدير.

**٣/ إختبار طبيعة البواقي:**

**١. رسم الاحتمالي الطبيعي: Normal Probability Plot**

من الشكل رقم (١) نلاحظ ان البواقي تتوزع طبيعياً وللتأكد من ذلك نقوم بالإختبار الأتي:

**بإختبار K-S Test لطبيعة البواقي:**

ومن الشكل (٢) نجدان القيمة الاحتمالية (P-Value) تساوي (0.150) وهي أكبر من قيمة مستوي المعنوية (٠.٠٥) عليه نقبل فرض العدم إي ان الأخطاء تتوزع طبيعياً.

**٢-٤ التنبؤ للطاقة الكهرومائية المنتجة بالقيقاواط للسنوات القادمة:**

**جدول رقم (٣) يوضح التنبؤات المستقبلية للطاقة الكهرومائية**

| السنوات | القيم المتنبأ بها |
|---|---|
| 2022 | 1956.11 |
| 2023 | 1990.55 |
| 2024 | 2025 |
| 2025 | 2059.47 |
| 2026 | 2093.94 |
| 2027 | 2128.43 |
| 2028 | 2162.93 |
| 2029 | 2197.44 |
| 2030 | 2231.96 |
| 2031 | 2266.5 |
| 2032 | 2301.04 |
| 2033 | 2335.6 |

**المصدر: إعداد الباحث بواسطة برنامج SPSS ٢٠٢٢م**

**٣. الإستنتاجات والتوصيات:**

**٣-١ الإستنتاجات:**

مما تقدم يمكن تلخيص النتائج التالية:

١. هنالك إستجابة غير خطية للتوليد المائي مع مرور الزمن.

٢. نموذج القوي (*Power Model*) هو النموذج الانسب في تمثيل الظاهرة مقارنة مع النماذج الاخري.

٣. النموذج الذي تم التوصل إليه كفؤ ويمكن الإعتماد عليه.

٤. يمكن إستخدام النموذج الذي توصل إليه البحث لمعرفة إتجاهات التوليد المائي من قبل الجهات التخطيطية لتحليل ودراسة الظاهرة.

**٣-٢ التوصيات:**

١. يمكن إستخدام النموذج الذي توصل إليه الباحث من قبل الجهه المستفيدة (الشركة السودانية للتوليد المائي) لمعرفة الإتجاهات المستقبلية للظاهرة ووضع الخطط اللازمه لها.

٢. يوصي الباحثان إستخدم تحليل السلاسل الزمنية وفق منهجية بوكس جنكيز كأسلوب آخر لدراسة ما ستؤول اليه الظاهرة مستقبلاً.

٣. كما نوصي بإجراء دراسة مقارنة بين النماذخ الغير خطية وتحليل السلاسل الزمنية لمعرفة أيهما في أدق في التنبؤ.

٤. يوصي الباحث بالقيام بدراسات وبحوث تأخذ بعين الإعتبار كمية المياه المستخدمة في الري و الطاقة المولدة لمعرفة الزيادة النسبية الحقيقة في الطاقة المولدة لأن بحثنا هذا أخذ واقع حال التوليد المائي دون النظر إلى كمية المياه.

٥. يوصي الباحث إستخدام تحليل السلاسل الزمنية لمتعدد المتغيرات (*Multivariate time series analysis*) وتحليل الانحدار المتعدد (*Multiple regression*), وذلك من خلال أخذ السلسلة لعدة متغيرات مثل تكلفة الإنتاج و سعر إنتاج الوحدة و هذا من شأنه أن يجعل التنبؤات أكثر دقة.

٦. الإستفادة من حصة البلاد من مياه النيل لإنشاء محطات وسدود لتوليد الطاقة الكهربائية.

**المصادر :**

اسامة ربيع امين سليمان_التحليل الإحصائي للبيانات بإستخدام برنامج *Minitab*_ قسم الإحصاء والرياضة والتامين_كلية التجارة(السادات)_ جامعة المنوفية.

٢. بسام يونس ابراهيم _ عادل موسي يونس _ حاجي أنمار أمين (٢٠٠٢) _ الاقتصاد القياسي _ دار عزة للنشر والتوزيع _ الخرطوم_ السودان.

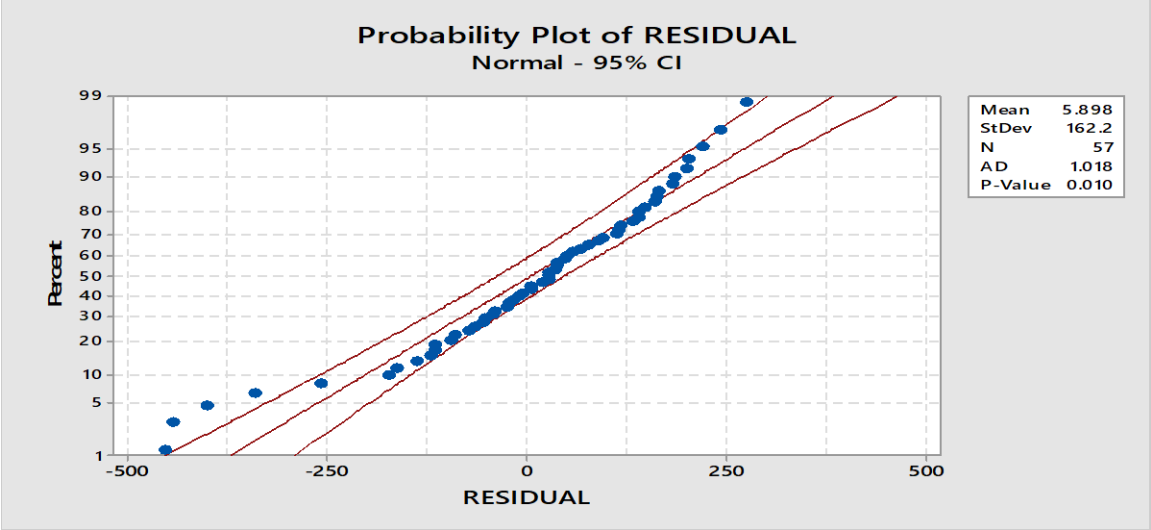
٣. عدنان عبد الرحمن ماجد (٢٠٠٢)، طرق التنبؤ الإحصائى، الجزء الأول، جامعة الملك سعود _ كلية العلوم _ قسم الإحصاء وبحوث العمليات.

٤. https://statisticsbyjim.com/regression/choose-linear-nonlinear-regression/.

٥. http://pzs.dstu.dp.ua/DataMining/mls/bibl/Nonlinear%20regression.pdf

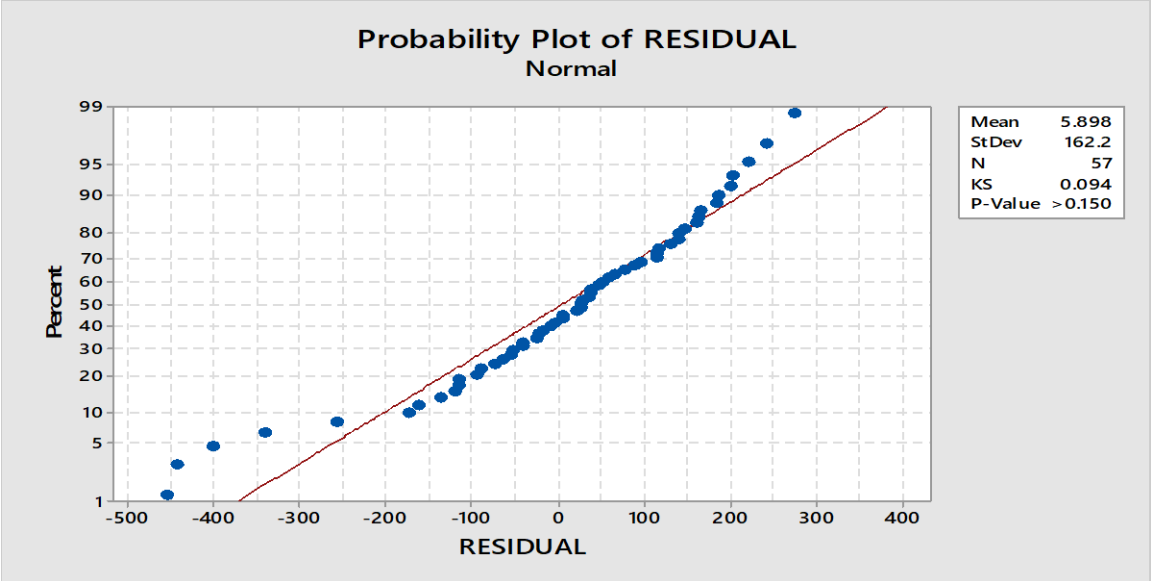
# الملحق

الشكل رقم (١) يوضح الاحتمال الطبيعي للبواقي



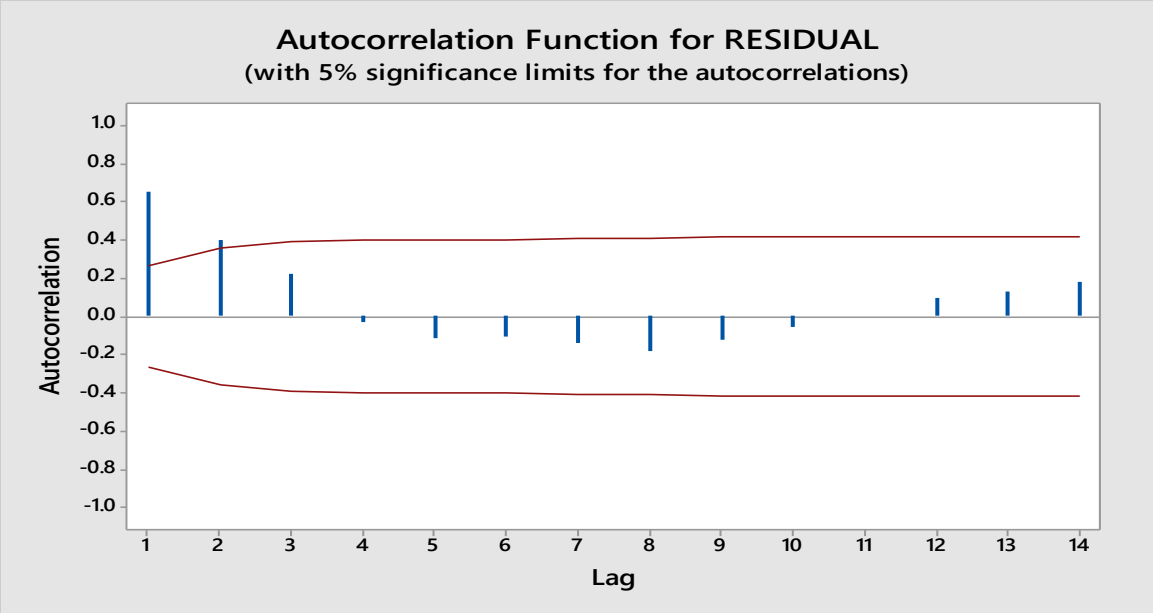

**المصدر: إعداد الباحث بواسطة برنامج Minitab ٢٠٢٢م**

الشكل رقم (٢) يوضح طبيعة البواقي



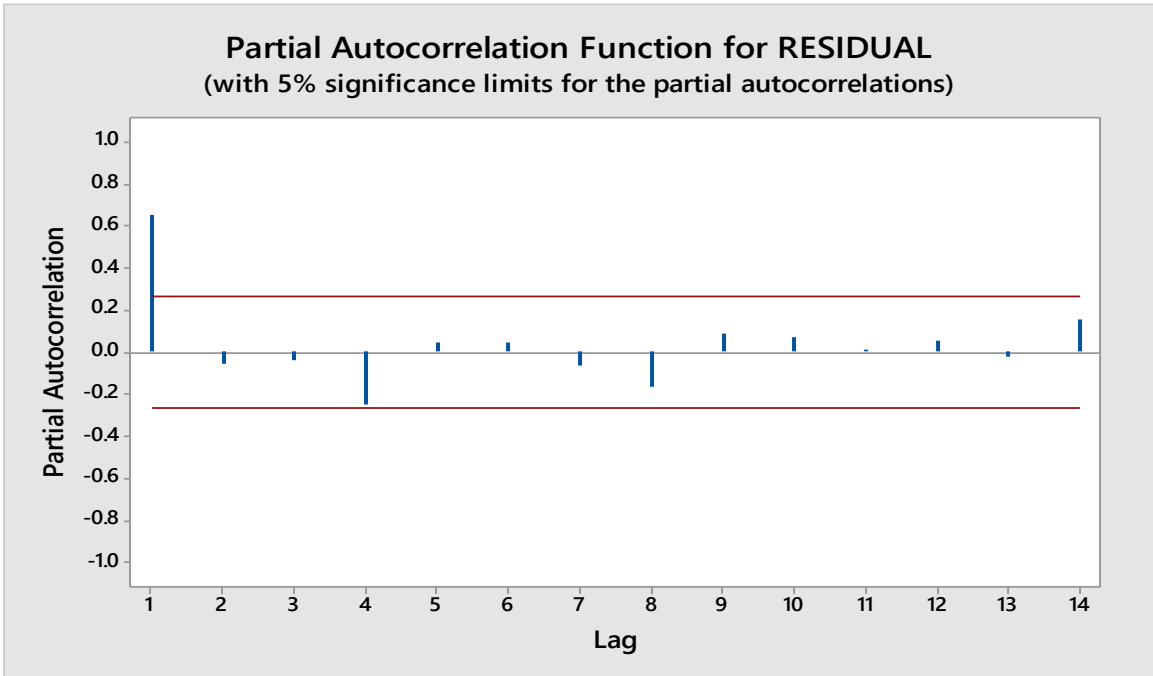

**المصدر: إعداد الباحث بواسطة برنامج Minitab ٢٠٢٢م**

**شكل رقم(٣) الـ ACF للبواقي**




المصدر: إعداد الباحث بواسطة برنامج Minitab ٢٠٢٢م

شكل رقم(٤) الـ PACF للبواقي




المصدر: إعداد الباحث بواسطة برنامج Minitab ٢٠٢٢

Research Article

# Toxic effects of mercury exposure on liver function test parameters in the traditional gold mining workers in Northern Sudan.

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## Abstract

The aim of this study is to evaluate the impact of mercury (Hg) exposure on liver function of the gold prospectors using mercury (Hg) for extraction of gold. It was descriptive cross sectional analytical one, carried out in Abu Hamad town in River Nile State – Northern Sudan from February to July 2018. It included eighty (80) participants, fifty (50) of them were gold prospectors as a case group and thirty (30) healthy individuals as control group.The result revealed that the average serum mercury level was significantly higher in the case group (63.6 µg/l) compared control group (3.0 µg/l) with *P*. value of (0.002). There was significant increase in serum transaminases .The average GGT in the case group was (38.0 IU/L) compared with (18.9 IU/L) of control group with *P*. value of (0.000). The level of the serum ALP in the case group was (82.8 IU/L) compared with (63.1 IU/L) of the control group with *P*. value of (0.000) . More alarming, result point to significant decrease in the structural function of the liver as reflected in the mean blood albumin in the case group of (3.9 g/dL) compared to (4.7 g/dL) of the control group with *P*. value of (0.000). There were insignificant differences in the other liver parameters between the case and the control group. There was a negative correlation between serum albumin and serum mercury level with r = (-0.23), with *P*.value of (0.04), and a positive correlation with serum.GGT r = (0.526), with *P*. value of (0.000).

**Keywords:** ***Mercury (Hg), Gold. Liver function test (LFTs), GGT, ALP*** **.**

## Introduction

The liver is an accessory digestive gland inhuman, detoxifies, various metabolites, synthesizes proteins, and produces biochemical molecules necessary for digestion . .A human liver, the largest gland in the body, normally weighs (1.44–1.66 kg)[1] .

The liver is highly specialized tissue with total number of functions of five hundred (500).The four major functions are excretion/secretion, synthesis, detoxification, and storage of various biomolecules[2] .

The most common liver function test (LFTs) include the serum aminotransferases such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), alkaline phosphatase, bilirubin, albumin, and prothrombin time. Aminotransferases are intracellular hepatic enzymes that leak into the circulation and serve as a marker of hepatocyte injury. Alkaline phosphatase (ALP), gama-glutamyltranspeptidase (GGT), and bilirubin act as markers of biliary function and cholestasis. Albumin and prothrombin reflect liver synthetic function [3].

Mercury (Hg) is heavy, silvery-white liquid toxic naturally occurring metal, with atomic number of eighty (80). It exists naturally as elemental Hg, in inorganic mercurous and mercuric compounds, and in organic Hg compounds. [4,5] .

Most of the mercury in the atmosphere is elemental mercury vapor; most of the mercury in water, soil, plants, and animals is inorganic and organic[6] .

In cosmetics, Mercury, as thiomersal, is widely used in the manufacture of mascara[7] .

Historically, mercury was used extensively in hydraulic gold mining in order to help the gold to sink through the flowing water-gravel mixture. Thin gold particles may form mercury-gold amalgam and therefore increase the gold recovery rates[8,9] .

Routes of exposure include inhalation, ingestion , cutaneous, injection and dental amalgams.

Inhaled mercury vapor is retained in the lungs to about 80%, whereas liquid metallic mercury passes the gastrointestinal tract almost unabsorbed[10] .

Most of the dietary intake of mercury comes from consumption of meat and fish products[11].

The half-lives vary from five (5) days in blood for phenyl mercury to ninety (90 ) days in urine for chronic exposure to inorganic mercury and the half-life of total Hg in the blood was found to be fifty seven (57) days on average in a Japanese study[12,13] .

Mercury is removed from the human body through urine or feces. The highest accumulation of mercury is in kidney, liver, spleen, and brain. Mercury can accumulate in pituitary and thyroid glands, the pancreas, and the reproductive organs.

Movement of mercury across the blood-brain barrier appears to be dependent on coupling with the amino acid cystine[14].

The toxicity of high-level mercury exposure is well known from Minamata disease in Japan outbreaks of which occurred in 1956 and 1965 due to the consumption of mercury -contaminated seafood and in Iraq outbreak[12.15] .

In addition to neurological toxicity, high levels of mercury exposure affect various human organs, including the cardiovascular, endocrine, reproductive, and immune systems [16] . The mechanisms of its toxicity have been suggested to involve degeneration, oxidative stress, and changes in the energy metabolism of the cell, but are not fully understood[17] .

Mercury toxicity is due to binding to protein with sulfhydryl and selenohydryl groups., which results in a change of its structure and function , that causes inhibition of many enzymes. Binding of mercury to intestinal proteins after ingestion of inorganic mercury results in acute gastrointestinal disturbances[18] .

With massive acute exposure to mercury vapor, erosive bronchitis and bronchiolitis potentially leading to respiratory failure may be accompanied by CNS symptoms such as tremor [19].

These symptoms may regress with cessation of exposure, but in many cases do not. Persistent neurological symptoms are common [20] .

**Rationale**

The rates of traditional random prospecting for gold using mercury for its extraction have increased dramatically in Sudan and in many other developing countries in Africa, Asia and South America. Most of the workers are less educated poor class of society. Exposure to mercury carried grave health hazards consequences in the absence of necessary safety precautions. There are few published studies, worldwide addressing this emerging situation. This study added value and fill the existing gap in the knowledge regarding the health of traditional gold mining workers .

**Objectives of the study**

The general objective of this study is to evaluate the effect of mercury exposure on liver function in gold prospectors using mercury in gold mining, while the specific objectives include:

1. To evaluate liver proteins (total protein and albumin) and liver enzymes (AST, ALT, ALP , GGT) in exposed individuals , in comparison to non exposed ones.
2. To measure mercury (Hg) concentration in exposed and non exposed individuals.

**Material and methods:**

The study was descriptive cross sectional analytical, carried out in Abu Hamad town in River Nile State – Northern Sudan from February to July 2018 to evaluate the effect of mercury exposure on liver function.

The study populations were gold mining workers as test group and other healthy individuals without exposure to mercury as control group.

Those who have liver disease or alcoholism are excluded from the study

Fifty (50) participants of gold mining workers were selected randomly as case group, and thirty (30) ones healthy volunteers with no exposure to mercury were selected randomly as control group.

Three (3) ml of venous blood samples were collected by standard procedure in vaccuated plain blood container, serum was separated and tested.

Structural interview questionnaire was filled, which contain name, age, duration of mercury exposure, history of liver disease and investigations results.

Total protein, albumin, Aspartate amino transferees (AST/GOT), Alanine amino transferase (ALT or GPT), Alkaline phosphatase (ALP), gama-glutamyltranspeptidase (GGT), were measured by standard method using full automated chemistry analyzer A15 Biosystem, employing a complete system of dedicated reagents for Clinical Chemistry and Turbidimetry designed to achieve the best possible performance, provide by BioSystems S.A. Costa Brava, 30. 08030 Barcelona (Spain). The results expressed as units/L.

Mercury (Hg) was measured by direct mercury analyzer (Milestone's DMA-80 Direct Mercury Analyzer ) is redefining mercury analysis.. The DMA-80 requires no sample preparation and delivers results, in as little as five (5) minutes. The results expressed as (µg/L).

**Statistical analysis**

All collected data was analyzed using statistical package for social sciences (SPSS), for windows, version 16.0. The data was represented as mean ± stander deviation .Mean values were compared using paired student t-test for calculating degree of variation, *P*. value<0.05 was considered as statistically significant. Analysis of variance (ANOVA) was used for continuous data and the statistical results were presented as means ± SEMs. Correlation and coefficient were also obtained.

**Ethical clearance**

The study was approved by research ethical committee of Faculty of Graduates Studies and Scientific Research in Shendi University. After explanation of the aims of the study, informed consent was obtained from each participant. There was full commitment precaution as regard sampling procedure, privacy and confidentiality.

**Result**

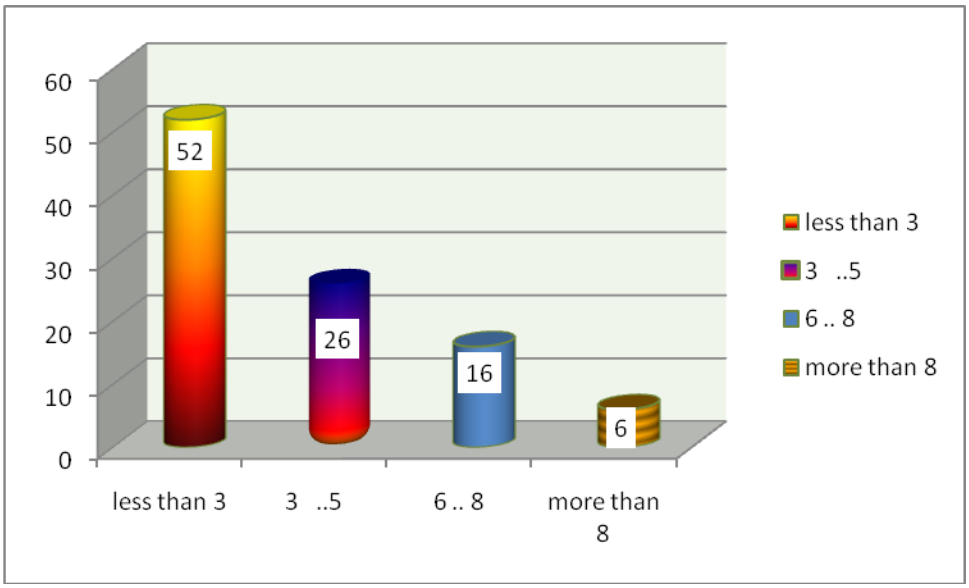



**Figure (1): Distribution of study group according to duration of mercury exposure in years .**

**Table (1): Comparison between serum total protein, albumin, ALP, AST, ALT, GGT and mercury concentrations in the test group and control.**

| Parameters | Frequency of control | Frequency of test | Mean of test | Mean of control | Sig.(2-tailed) |
|---|---|---|---|---|---|
| **Total protein** | 30 | 50 | 7.8 | 7.6 | 0.258 |
| **Albumin** | 30 | 50 | 3.9 | 4.7 | 0.000* |
| **ALP** | 30 | 50 | 82.8 | 63.1 | 0.000* |
| **AST** | 30 | 50 | 20.9 | 19.9 | 0.469 |
| **ALT** | 30 | 50 | 21.2 | 21.5 | 0.833 |
| **GGT** | 30 | 50 | 38.0 | 18.9 | 0.000* |
| **Mercury** | 30 | 50 | 63.6 | 3.0 | 0.002* |

Independent t- test

*Significant level is <0.05

**Table (2) Comparison between serum total protein, albumin, ALP, AST, ALT and GGT concentration in the test group in normal mercury level and high mercury level.**

| | Normal mercury level | | High mercury level | | |
|---|---|---|---|---|---|
| **Parameter** | **Frequency** | **Mean** | **Frequency** | **Mean** | **Sig.(2-tailed)** |
| **Total protein** | 12 | 7.8 | 38 | 7.8 | 0.603 |
| **Albumin** | 12 | 3.8 | 38 | 3.9 | 0.679 |
| **ALP** | 12 | 82.1 | 38 | 83.1 | 0.881 |
| **AST** | 12 | 23.1 | 38 | 20.9 | 0.160 |
| **ALT** | 12 | 20.2 | 38 | 21.5 | 0.607 |
| **GGT** | 12 | 19.8 | 38 | 43.8 | 0.000* |

Independent t- test

*Significant level is <0.05

**Table (3): Comparison between mean concentration of serum total protein, albumin, ALP, AST, ALT, GGT, mercury and the duration of mercury exposure.**

| **Parameter** | **Duration of exposure** | **Frequency** | **Mean** | ***P*. value** |
|---|---|---|---|---|
| **Total protein** | Less than 3 years | 26 | 7.7 | 0.093 |
| | 3 - 5 years | 13 | 7.9 | |
| | 6 - 8years | 8 | 8.1 | |
| | More than 8 years | 3 | 7.6 | |
| **Albumin** | Less than 3 years | 26 | 3.8 | 0.197 |
| | 3 - 5 years | 13 | 3.9 | |
| | 6 - 8years | 8 | 4.0 | |
| | More than 8 years | 3 | 3.6 | |
| **ALP** | Less than 3 years | 26 | 82.1 | 0.690 |
| | 3 - 5 years | 13 | 81.7 | |
| | 6 - 8years | 8 | 89.7 | |
| | More than 8 years | 3 | 75.6 | |
| **AST** | Less than 3 years | 26 | 19.5 | 0.121 |
| | 3 - 5 years | 13 | 22.1 | |
| | 6 - 8years | 8 | 24.7 | |
| | More than 8 years | 3 | 17.6 | |

| | | | | |
|---|---|---|---|---|
| **ALT** | Less than 3 years | 26 | 21.4 | 0.497 |
| | 3 - 5 years | 13 | 19.5 | |
| | 6 - 8years | 8 | 24.1 | |
| | More than 8 years | 3 | 19.0 | |
| **GGT** | Less than 3 years | 26 | 36.0 | 0.054 |
| | 3 - 5 years | 13 | 32.9 | |
| | 6 - 8years | 8 | 54.5 | |
| | More than 8 years | 3 | 34.0 | |
| **Mercury** | Less than 3 years | 26 | 83.9 | 0.549 |
| | 3 - 5 years | 13 | 43.3 | |
| | 6 - 8years | 8 | 45.5 | |
| | More than 8 years | 3 | 23.8 | |

One way ANOVA

*Significant level is <0.05

**Table (4) Compression between mean concentration serum total protein, albumin, ALP, AST, ALT, GGT, mercury and age:**

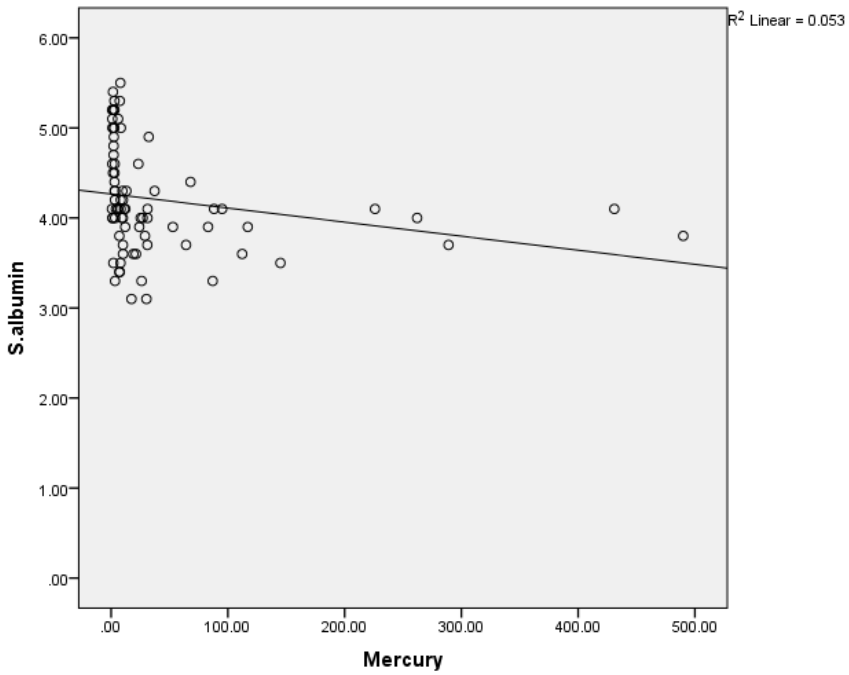
| **Parameter** | **Age** | **Frequency** | **Mean** | ***P*. value** |
|---|---|---|---|---|
| **Total protein** | Less than 20 years | 4 | 7.7 | 0.813 |
| | 20 - 30 years | 30 | 7.8 | |
| | 31 - 40 years | 9 | 7.8 | |
| | More than 40 years | 7 | 7.9 | |
| **Albumin** | Less than 20 years | 4 | 3.8 | 0.665 |
| | 20 - 30 years | 30 | 3.9 | |
| | 31 - 40 years | 9 | 3.9 | |
| | More than 40 years | 7 | 3.9 | |
| **ALP** | Less than 20 years | 4 | 83.7 | 0.718 |
| | 20 - 30 years | 30 | 81.1 | |
| | 31 - 40 years | 9 | 89.5 | |
| | More than 40 years | 7 | 81.0 | |
| **AST** | Less than 20 years | 4 | 18.2 | 0.441 |
| | 20 - 30 years | 30 | 20.2 | |
| | 31 - 40 years | 9 | 23.4 | |
| | More than 40 years | 7 | 21.8 | |
| **ALT** | Less than 20 years | 4 | 22.2 | |
| | 20 - 30 years | 30 | 21.5 | |

| | | | | |
|---|---|---|---|---|
| | 31 - 40 years | 9 | 21.8 | 0.719 |
| | More than 40 years | 7 | 18.3 | |
| **GGT** | Less than 20 years | 4 | 29.2 | 0.057 |
| | 20 - 30 years | 30 | 35.1 | |
| | 31 - 40 years | 9 | 53.1 | |
| | More than 40 years | 7 | 36.3 | |
| **Mercury** | Less than 20 years | 4 | 42.7 | 0.679 |
| | 20 - 30 years | 30 | 70.1 | |
| | 31 - 40 years | 9 | 81.9 | |
| | More than 40 years | 7 | 24.1 | |

One way ANOVA

*Significant level is <0.05

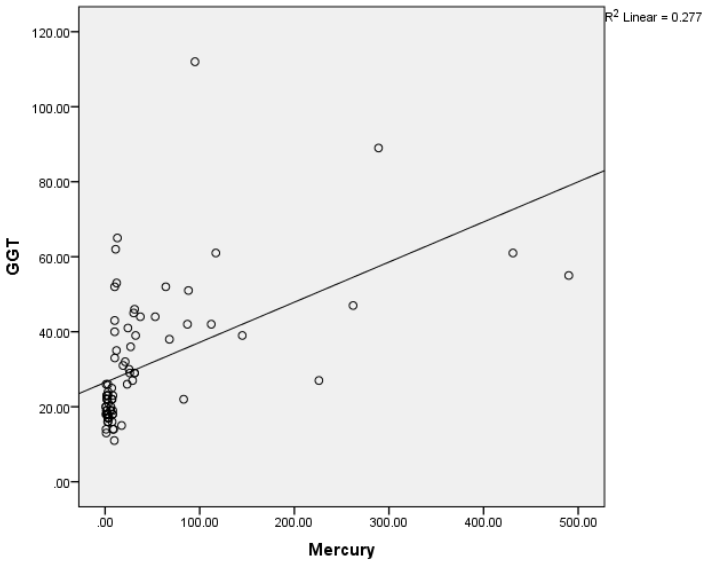
**Figure (2): Relationship between mercury and albumin concentration**




Personal correlation -0.23

*P*. value 0.04

**Figure (3): Relationship between mercury and GGT concentration.**




Personal correlation 0.526

*P*. value 0.000

## Discussion

The current study was carried out to evaluate the impact of mercury exposure on liver function among gold mining workers in Abu Hamad town, River Nile state in Northern Sudan from February to July 2018.

Eighty (80) blood samples were collected, Fifty (50) of these samples were collected from gold mining workers as test group, and thirty (30) samples were collected from health individual as control group.

The result of this study revealed that the average serum mercury level was significantly higher in the case group (63.6 µg/) compared to control group (3.0 µg) with *P*. value of (0.002). Similar findings noticed in the study conducted in the same area by Eltayeb Tayrab, and his colleagues from December 2012 to January 2014, and revealed that the serum mercury in the traditional gold miners was (24.9 ± 32.24mg/l) versus (1.40 ± 0.94mg/l) in the non-exposed control group; with P value (p= 0.000) [(21)] . Similar result was also stated by

Babak Mostafazadeh, et al. who found that the mean serum mercury levels were 208.14 µg/L from gold mining workers in the northwest of Iran, who also had associated neurotoxity [(22)]. Lina Eltaib ,et al also repotted consistent findings of serum mercury concentrations exceed concentration of (100 ug/L) in 98% of exposed gold miners from Altwahen near Abu-Hamed, in Northern Sudan, the same area of the current study[(23)].

Statistical analysis of results revealed that, there was significant increase in serum GGT, mean serum GGT in test group was (38.0 IU/L) when compared with control group was (18.9 IU/L) with *P*. value of 0.000, and significant increase in serum level of ALP in test group was (82.8 IU/L) when compared with control group of (63.1IU/L) with *P*. value of (0.000), and there was significant decrease in synthetic function of liver because the mean of serum albumin in test group was (3.9

g/dL) when compared with control group was (4.7 g/dL) with *P*. value of (0.000), there was insignificant differences in other liver function parameters between the test and control group. The mean level of serum mercury in test group was (63.6) and in control group was (3.0) with *P*. value of (0.002).

The study also demonstrated significant effect of mercury level on serum GGT because the mean of GGT in group of normal mercury level was (19.8IU/L) when compared with pathologic level of mercury was (43.8IU/L) with *P*. value of (0.000), that mean the mercury had significant effect on serum GGT level.

This study findings consistent with those obtained by [24] . in Minamata area in Japan. The findings of this study was also supported those presented in the CASPIAN III study[25].

There was negative correlation between serum albumin and serum mercury level because r= (-0.23), with *P*. value of (0.04), and positive correlation with S.GGT r= (0.526), with *P*. value of (0.000). The present study results proved that there was insignificant effect of the age and duration of mercury exposure on liver function test parameters, with *P*. value ( > 0.05).

The toxic effect of mercury (Hg) exposure is not restricted to the liver, but it is multisystem involvement such as renal& respiratory impairment, neurotoxicity, sexual &endocrines malfunctions, skin manifestation and genetic toxicity [23,26].

The heavy seasonal rain in Northern Sudan in recent year 2021and 2022 could wash mercury into the Nile River, that led to contamination of Nile water and fishes

**Conclusion:**

On the basis of the current study findings, it was concluded that mercury (Hg) exposure had serious impact on liver function with associated elevation of serum concentration of GGT and ALP, and declining serum concentration of albumin

**Recommendations**

Since extraction of gold by mercury in recent years gaining increased popularity in Sudan and other developing countries, as source of income, driven by harsh economic conditions, with many thousands engaged in traditional gold mining. It's highly recommended, that those vulnerable important society group being educated about risks of unsafe use of mercury and alerted to the safe disposal of mercury-contaminated waste [27].

Use of personal protective equipments (PPEs) by workers should be adopted through all gold extraction operations. Establishment and governmental support of local health care facilities in concerned area to provide the proper care for the workers should be considered as

urgent priority. Further studies are urgently needed to evaluate mercury contamination of Nile River and underground water, and the carcinogenic and teratogenic effect on local community.

More important, the outlaws of mercury, cyanide in mining by Sudan cabinet at its periodical meeting in October 9th - 2019 in Khartoum, headed by Prime Minster Hamdok should be adopted .

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**Conflict of interest**

No conflict of interest to be declared by authors

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## Short Notes

# On equatorial characterization of zonoids and intersection bodies

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## Abstract

This study aimed to prove that there is no local equatorial characterization of zonoids in odd dimensions, this gives a negative answer to the conjecture posed by W. Weil in 1977 and shows that the local equatorial characterization of zonoids may be given only in even dimensions. In addition we prove a similar result for intersection bodies, we used the descriptive- deductive method, the study found that there is no local characterization of these bodies.

**Keywords**: *Equatorial characterization, zonoids, intersection bodies*

**المستخلص**

هدفت هذه الدراسة إلى إثبات إنه ليس هناك معيارللتساوى الموضعى للزونويد فى الأبعاد الفردية، هذا يعطى إجابة سلبية للتخمين الذى طرحه و.ويبل فى ١٩٧٧ و يبين إن المعيار للتساوى الموضعى للزونويد يمكن أن يعطى فقط فى حالة الأبعاد الزوجية. بالإضافة إلى إننا أثبتنا نتيجة مماثلة للأجسام المتقاطعة، إستخدمنا المنهج الوصفى- الإستنباطى، توصلت الدراسة إلى إنه ليس هناك معيار موضعى لهذه الإجسام.

**كلمات مفتاحية:** معيار تساوى ، زونويد ، أجسام متقاطعة

## Introduction

A zonoid in $\mathbb{R}^n$ is an origin symmetric convex body that can be approximated (in the Hausdorff metric) by finite Minkowski sums of line segments. It turns out that zonoids appear in many different contexts in convex geometry, physics, optimal control theory, and functional analysis (see [1], [3], [4]). One of the equivalent definitions of zonoids, useful in convex geometry, leads to

a notion of a projection body. An origin symmetric convex body $L$ in $\mathbb{R}^n$ is called a projection body is there exists another origin symmetric convex body $K$ such that the support function of $L$ in every direction is equal to the volume of the hyperplane projection of $K$ orthogonal to this direction: for every

$$\lambda \in T^{n-1}, h_L(\lambda) = \mathrm{Vol}_{n-1}(K|\lambda^{\perp}), \lambda^{\perp} = \{y \in \mathbb{R}^n : \lambda . y = 0\}.$$

The support function $h_L(\lambda) = \max_{x \in L} \lambda . x$ is equal to the dual norm $\|\lambda\|_{L^*}$ where $L^*$ stands for the polar body of $L$. From the above definition and Cauchy formula (see [15]), we immediately derive the following analytic definition, which will be useful for us in this study: An origin symmetric convex body $L \subset \mathbb{R}^n$ is a zonoid if and only if

$$h_L(\lambda) = Cos\mu(\lambda) \coloneqq \int_{T^{n-1}} |\lambda . \theta| d\mu(\theta)$$

with some even positive measure $\mu$ on $T^{n-1}$. Finally, a functional analytic definition shows that an origin symmetric convex body $L \subset \mathbb{R}^n$ is a zonoid if and only if it is a polar body to the unit ball of a subspace of $L_1$.

It is well known that every origin symmetric convex body in $\mathbb{R}^n$ is projection body, but this is no longer true in $\mathbb{R}^n$ for $n \geq 3$ (see [26],

[15]). It is an interesting question how to determine if a given convex body is a zonoid or not. It is very reasonable to assume that one can provide a strictly local characterization of zonoids. This question was posed repeatedly (see [26] , for the history of problem), however [28] showed that a local characterization of zonoids does not exist. In particular, he showed that there exists an origin symmetric convex $C^{\infty}$ body $K \subset \mathbb{R}^n, n \geq 3$, that is not a zonoid but has the following property: for every $u \in T^{n-1}$ there exists a zonoid $Z_u$ centered at the origin and a neighborhood $U_u \subset T^{n-1}$ of $u$ such that the boundaries of $K$ and $Z_u$ coincide at all points where the exterior unit normal vectors belong to $U_u$. Thus, no characterization of zonoids that involves only arbitrarily small neighborhoods of boundary points is possible.(see [28]) ,Weil proposed the following conjecture about local equatorial characterization of zonoids. Let $L \subset \mathbb{R}^n$ be an origin symmetric convex body and assume that for any equator $\varphi \subset T^{n-1}$, there exists a zonoid $Z_{\varphi}$ and a neighborhood $E_{\varphi}$ of $\varphi$ such that the boundaries of $L$ and $Z_{\varphi}$ coincide at all points where the exterior unit vector belongs to

$E_\varphi$ ; then $L$ is a zonoid. Affirmative answers for even dimensions were given independently by [23], [13] but the question was left open in odd dimensions. That was a consequence of the fact that the inversion formulas for the cosine transform are not local in odd dimensions (see [22]).

In this study we show that the answer to the conjecture in odd dimensions is negative. We prove that in both cases (for odd and even dimensions) the answer can be obtained as a consequence of the characterization of zonoids in terms of sections of the polar body, given in [18]. In even dimensions the answer follows directly from the geometric inversion formula for Cosine transform [18]. The odd dimensional case,on the other hand, requires much more tricky and detailed analysis of the behavior of the inverse Cosine transform.

Our main tool is the Fourier analytic inversion formula from [8] or [15]. It allows to obtain the results for zonoids together with the results about the intersection bodies. The notion of an intersection body of star body was introduced by [21]. $K$ is called the intersection body of $L$ if the radius of $K$ in every direction is equal to the $(n-1)$–dimensional volume of the central hyperplane section of $L$ perpendicular to this direction: $\forall \lambda \in T^{n-1}$

$$\rho_k(\lambda) = \mathrm{Vol}_{n-1}(L \cap \lambda^{\perp}), \lambda^{\perp},$$

where $\rho_k(\lambda) = \max\{a: a\lambda \in K\}$ is the radial function of body $K$. Passing to polar coordinates in $\lambda^{\perp}$, we derive the following analytic definition of an intersection body of star body: $K$ is called the intersection body of $L$ if

$$\rho_k(\lambda) = \frac{1}{n-1}\psi\rho_L^{n-1}(\lambda)$$
$$:= \frac{1}{n-1}\int_{T^{n-1}\cap\lambda^{\perp}} \rho_L^{n-1}(\theta)d\theta.$$

Here $\psi$ stands for the spherical Radon transform (see [22]).

A more general class of intersection bodies was defined by ([5] and [29] as the closure of intersection bodies of star bodies in the radial metric

$$d(K,L) = \sup_{\lambda\in T^{n-1}}|\rho_k(\lambda) - \rho_L(\lambda)|.$$

In this study we will consider only $C^{\infty}$ smooth intersection bodies: a body $K$ is an intersection body if there exists an even nonnegative function $f$ on $T^{n-1}$, such that the radial function of $K$ is a spherical Radon transform $\psi f$ of $f$. Since we can always define

$L: \rho_L^{n-1}(\theta) = (n-1)f(\theta)$,

we will not distinguish between intersection bodies of star bodies and intersection bodies. We prove that the local equatorial characterization of intersection bodies is not possible in odd dimensions. Namely, we show that one can construct an origin symmetric convex body $L \subset \mathbb{R}^n, n \geq 5$ is odd, such that for any equator

$\varphi \subset T^{n-1}$, there exists an intersection body $I_\varphi$ and a neighborhood $E_\varphi$ of $\varphi$ such that the boundaries of $L$ and $I_\varphi$ coincide at all points of $E_\varphi$ (i.e, $\rho_L(\lambda) = \rho_{I_\varphi}(\lambda)$ for all $\lambda \in E_\varphi$); but nevertheless, $L$ is not an intersection body. On the other hand, we show that the local equatorial characterization of intersection bodies is possible in even dimensions.

We also extend the result of [28] to the class of intersection bodies by proving that there is no local characterization of those bodies in odd and even dimensions.We prove that there exists an origin symmetric convex $C^\infty$ body $K \subset \mathbb{R}^n, n \geq 5$, that is not an intersection body, but has the following property : for each $u \in T^{n-1}$ there exists an intersection body $I_u$ centered at the origin and a neighborhood $U_u \subset T^{n-1}$ of $u$ such that the boundaries of $K$ and $I_u$ coincide on $U_u$. In odd dimensions this is a consequence of the lack of a local equatorial characterization of intersection bodies mentioned above but we give an independent proof that does not distinguish between even and odd dimensions.

Our proofs for zonoids and intersection bodies are very similar, they are based on almost identical Fourier analytic inversion formulas for the Cosine and Radon transforms. This is one more indication of the remarkable duality between sections and projections (see[19]) .

## 1. Auxiliary results

Our main tool is the Fourier transform of distributions (see [9],[10] and [15] for exact definitions and properties) and the connections between the Cosine and the spherical Radon transforms and the Fourier transform.

We start with the connection of the spherical Radon transform and the Fourier transform. A Koldobsky (see for example Lemma 3.7 in [15]) proved that

$$\psi g(\lambda) = \frac{1}{\pi}\hat{g}(\lambda), \qquad \forall \lambda \in T^{n-1}, \qquad (1)$$

provided that $g$ is an even homogeneous function of degree $-n+1$ on $\frac{\mathbb{R}^n}{\{0\}}.n>1$, satisfying $g|_{T^{n-1}} \in L_1(T^{n-1})$.

An immediate consequene of this formula is the following Fourier analytic a characterization of intersection bodies (see [15] Theorem 4.1): An origin symmetric star body $K$ is an intersection body if and only if $\rho_K$, extended to $\mathbb{R}^n$ as a homogeneous function of degree $-1$, represents a positive definite distribution on $\mathbb{R}^n$. When $K$ is infinitely smooth, this is equivalent to $\widehat{\rho_K} \geq 0$.

A very similar connection of the Cosine transform and the Fourier transform was established in ([18] see also [15]):

$$Cosg(\lambda) = \frac{2}{\pi}\hat{g}(\lambda), \qquad \forall \lambda \in T^{n-1}, \qquad (2)$$

provided that $g$ is an even homogeneous function of degree $-n-1$

on $\frac{\mathbb{R}^n}{\{0\}}.n>1$, satisfying $g|_{T^{n-1}} \in L_1(T^{n-1})$.

As above, one can obtain a very similar Fourier analytic a characterization of zonoids (see [15] Theorem 8.6): An origin symmetric star body $K$ is a zonoid if and only if $h_K$, extended to $\mathbb{R}^n$ as homogeneous function of degree 1, represents a negative definite distribution on $\mathbb{R}^n$. When $K$ is infinitely smooth, this is equivalent to $\widehat{h_K} \leq 0$.

Our next tool is a formula connecting the Fourier transform of powers of the radial function with the derivatives of the parallel section function. Let $D$ be an infinitely smooth origin symmetric star body in $\mathbb{R}^n, \lambda \in T^{n-1}$, and let

$\lambda^{\perp} = \{x \in \mathbb{R}^n : x.\lambda = 0\}$. We denote by

$$A_{D,\lambda}(s) = \mathrm{Vol}_{n-1}(D \cap \{\lambda^{\perp} + s\lambda\}), s \in \mathbb{R},$$

The parallel section function of $D$ in the direction of $\lambda$. The following formula was proved in [8] see [15]: for any $\lambda \in T^{n-1}$ and $k \in \mathbb{N}, k \neq n-1$,

$$\widehat{\rho_D^{n-k-1}}(\lambda) = (-1)^{\frac{k+1}{2}}\pi(n-k-1)A_{D,\lambda}^{k}(0), \qquad (3)$$

when $k$ is even, and

$$\widehat{\rho_D^{n-k-1}}(\lambda) = (-1)^{\frac{k+1}{2}}2(n-k-1)k!\int_0^{\infty}\frac{A_{D,\lambda}(z) - A_{D,\lambda}(0) - \cdots - A_{D,\lambda}^{(k-1)}(0)\frac{z^{k+1}}{(k-1)!}}{z^{k+1}}dz, \qquad (4)$$

when $k$ is odd.

As a consequence of equations (1), (3) and (4) with $k = n-2$, we obtain the

Fourier analytic a characterization of intersection bodies ( see [15]).

Let $L$ be an origin symmetric star body in $\mathbb{R}^n$ such that $\rho_L$ is infinitely differentiable on $\lambda \in T^{n-1}$. The body $L$ is an intersection body if and only if $\forall \lambda \in T^{n-1}$,

$$(-1)^{\frac{n-2}{2}} A_{L,\lambda}^{(n-2)}(0) \geq 0, \qquad (5)$$

when $n$ is even, and

$$(-1)^{\frac{n-1}{2}} \int_0^{\infty} \frac{A_{L,\lambda}(z) - A_{L,\lambda}(0) - \cdots - A_{L,\lambda}^{(n-3)}(0)\frac{z^{n-3}}{(n-3)!}}{z^{n-1}} dz \geq 0, \qquad (6)$$

when $n$ is odd.

Similarly, using the duality relation $h_D = \rho_{D^*}^{-1}$ and equations (2), (3) and (4) with $k = n$, one can obtain the following characterization of of zonoids (see [18] or [15]). Let $L$ be an origin symmetric convex body in $\mathbb{R}^n$ such that $h_L$ is infinitely differentiable on $T^{n-1}$. The body $L$ is a zonoid (projection body) if and only if $\forall \lambda \in T^{n-1}$,

$$(-1)^{\frac{n}{2}} A_{L^*,\lambda}^{(n)}(0) \geq 0, \qquad (7)$$

when $n$ is even, and

$$(-1)^{\frac{n+1}{2}} \int_0^{\infty} \frac{A_{L^*,\lambda}(z) - A_{L^*,\lambda}(0) - \cdots - A_{L^*,\lambda}^{(n-1)}(0)\frac{z^{n-1}}{(n-1)!}}{z^{n+1}} dz \geq 0, \qquad (8)$$

when $n$ is odd.

**2. There is no local equatorial characterization of intersection bodies in odd dimensions.**

To construct counterexample, it is natural to use (6). This formula shows that one has to use the information about the section function $A_{L,\lambda}(z)$ of the body along the whole range of $z$. For $0 < \epsilon < 1$ and $\lambda \in T^{n-1}$, we denote by $U_\epsilon(\lambda)$ the union of caps centered at $\lambda$ and $-\lambda$:

$$U_\epsilon(\lambda) := \left\{\theta \in T^{n-1} : |\theta.\lambda| \geq \sqrt{1-\epsilon^2}\right\}.$$

We denote by $E_\epsilon(\lambda)$, $0 < \epsilon < 1$, the neighborhood of the equator $T^{n-1} \cap \lambda^{\perp}$:

$$E_\epsilon(\lambda) := \{\theta \in T^{n-1} : |\theta.\lambda| < \epsilon\}.$$

The following result is crucial for the construction of the counterexample. Its proof is based on the fact that the inversion formula (6) is not local.

**Lemma 3.1.** Let $n \geq 3$ be odd. Then there exists an $\epsilon > 0$ and an absolute constant $c > 0$ such that for any $x, \lambda \in T^{n-1}$, there exists an even function $f_{x,\lambda}$ satisfying $f_{x,\lambda} = 0$ on $E_\epsilon(x)$, and

$$\psi^{-1} f_{x,\lambda} > c \text{ on } U_\epsilon(\lambda).$$

**Proof.** First, we fix $x, \lambda \in T^{n-1}$ and find $\epsilon = \epsilon(x,\lambda)$ and $c = c(x,\lambda)$ satisfying the

requirement of the lemma. Then we use the compactness argument to produce absolute $\epsilon$ and $c$.

For fixed $x, \lambda \in T^{n-1}$ and some small $\epsilon > 0$ we take two auxiliary infinitely smooth symmetric star bodies $M, Q$, such that $\rho_M = \rho_Q$ on the closure of $E_\epsilon(\lambda) \cup E_\epsilon(x)$, and $\rho_M > \rho_Q$ otherwise. We put $f_{x,\lambda} = (-1)^{\frac{n-1}{2}}(\rho_M - \rho_Q)$. Then $f_{x,\lambda} = 0$ on $E_\epsilon(x)$, and $\rho_M = \rho_Q$ on $E_\epsilon(\lambda)$ implies

$$A^k_{M,\lambda}(0) = A^k_{Q,\lambda}(0), k = 0,1, \dots, n-3.$$

Thus (1) and (4) with $k = n-2$ imply

$$\psi^{-1} f_{x,\lambda}(\lambda) = (-1)^{\frac{n-1}{2}} \left(\psi^{-1}\rho_M(\lambda) - \psi^{-1}\rho_Q(\lambda)\right) = (-1)^{n-1}(2\pi)^{n-1}(n-2)! \int_0^\infty \frac{A_{M,\lambda}(z) - A_{Q,\lambda}(0)}{z^{n-1}} dz > 0,$$

since $Q \subseteq M$. We proved that for fixed $x, \lambda \in T^{n-1}$ there exists $\epsilon' = \epsilon'(x,\lambda) > 0$ and $c' = c'(x,\lambda) > 0$ such that there exists an even function $f_{x,\lambda}$ satisfying $f_{x,\lambda} = 0$ on $E_\epsilon(x)$, and $\psi^{-1} f_{x,\lambda}(\lambda) \geq c'$. The function $\psi^{-1} f_{x,\lambda}$ is continuous on $T^{n-1}$ since $M, Q$ are infinitely smooth (see [15] Lemma 2.4). Hence,

$\psi^{-1} f_{x,\lambda} \geq c > 0$ on $U_{\epsilon''}(\lambda)$, for some $\epsilon'' > 0$ and $c = c(x,\lambda)$.

Put $\tilde{\epsilon} = \tilde{\epsilon}(x,\lambda) = \min(\epsilon', \epsilon'')$. We prove that for any $x$ and $\lambda$, there is

$\tilde{\epsilon} = \tilde{\epsilon}(x,\lambda) > 0$ and a function $f_{x,\lambda}$ such that $f_{x,\lambda} = 0$ on $E_{\tilde{\epsilon}}(x)$, but $\psi^{-1} f_{x,\lambda} \geq c$ on $U_{\tilde{\epsilon}}(\lambda), c = c(x,\lambda)$.

Now we use the compactness argument to show that we can choose $\tilde{\epsilon}$ and $c$ independent of $x$ and $\lambda$. We choose a finite set of pairs $\{x_i, \lambda_i\}_{i=1}^m$ such that $\left\{U_{\frac{\tilde{\epsilon}}{2}}(x_i) \times U_{\frac{\tilde{\epsilon}}{2}}(\lambda_i)\right\}_{i=1}^m$ cover $T^{n-1} \times T^{n-1}$. We take

$$\epsilon = \frac{1}{2} \min_{1 \leq i \leq m} \tilde{\epsilon}_i \text{ and } c = \min_{1 \leq i \leq m} c(x_i, \lambda_i).$$

Then, for any $(x,\lambda)$, there is a pair $(x_i, \lambda_i)$ such that

$(x,\lambda) \in U_{\frac{\tilde{\epsilon}}{2}}(x_i) \times U_{\frac{\tilde{\epsilon}}{2}}(\lambda_i)$ and thereby

$$E_\epsilon(x) \times U_\epsilon(\lambda) \subset E_{\tilde{\epsilon}_i}(x_i) \times U_{\tilde{\epsilon}_i}(\lambda_i).$$

Finally, we may define $f_{x,\lambda} = f_{x_i,\lambda_i}$ (see [22]).

**Remark 3.2.** Note that, dilating $M$ and $Q$ (and thus functions $f_{x,\lambda}$), we may assume that $c$ is as large as we want. By the technical resons that will become clear later, we take $c = 2\psi^{-1}1$. Moreover, we can assume that the set of functions $\{f_{x,\lambda}\}_{x,\lambda \in T^{n-1}}$ in the lemma is finite.

Let $C_+^\infty$ be the class of origin symmetric convex bodies with $C^\infty$ boundary and everywhere positive Gaussian curvature (see [6]). The following auxiliary result seems to be well-known. It is interesting to note that it is not true without the $C_+^\infty$ assumption though (see [26] [14], [20], [2]).

**Lemma 3.3.** Let $M \in C_+^\infty$ and let

$K(s) = sB_2^n + (1-s)M$ be Minkowski sum of $sB_2^n$ and $(1-s)M, s \in [0,1]$. Then the map

$s \to \psi^{-1}\rho_{k(s)}(\lambda), \lambda \in T^{n-1}$, is continuous.

**Proof.** We note first that for any fixed $s \in [0,1]$, the boundary $\partial K(s)$ of $K(s)$ is $C^\infty$. Indeed, $\partial K(s)$ can be parameterized as

$$u \in T^{n-1} \to \nabla h_{(1-s)M}(u) + su = (1-s)\nabla h_M(u) + su,$$

where $u \in T^{n-1} \to (1-s)\nabla h_M(u)$

is a parameterization of $(1-s)\partial M$. Here

$$\nabla h_{(1-s)M}(u) = v^{-1}(u),$$

and $v: (1-s)\partial M \to T^{n-1}$

is the spherical image map (see [6] ,[26]), to show that the map $u \in T^{n-1} \to \nabla h_M(u)$ is a $C^\infty$ diffeomorphism. Hence, the map

$u \in T^{n-1} \to g_s(u) := (1-s)\nabla h_M(u) + su$

is also a $C^\infty$ diffeomorphism. To show that $s \to \psi^{-1}\rho_{k(s)}(\lambda)$ is continuous, we pick any $s \in [0,1]$ and take any sequence $\{s_m\}_{m=1}^\infty$ of points from $[0,1]$ converging to $s$. The map

$$u \in T^{n-1} \to f_s(u) := \frac{g_s(u)}{|g_s(u)|}$$

is a $C^\infty$ diffeomorphism for any $s \in [0,1]$, and $f_{s_m} \to f_s$ in $C^\infty(T^{n-1})$. Hence, $f_{s_m}^{-1} \to f_s^{-1}$ in $C^\infty(T^{n-1})$.

Now, $g_s\left(f_s^{-1}(\lambda)\right) \in \partial K(s)$ implies $\rho_{k(s)}(\lambda) = \left|g_s\left(f_s^{-1}(\lambda)\right)\right|$, and $\rho_{k(s_m)}$ converges to $\rho_{k(s)}$ in $C^\infty(T^{n-1})$. Since $\psi$ is a continuous bijection of $C^\infty(T^{n-1})$ to itself, (see [6]), the lemma is proved.

**Lemma 3.4.** Let $n \geq 5$. For any point $\lambda_0 \in T^{n-1}$ there exists $\widetilde{K} \in C_+^\infty$ such that $\psi^{-1}\rho_{\widetilde{K}}(\lambda)$ is strictly positive for all

$\lambda \neq \pm\lambda_0$ and $\psi^{-1}\rho_{\widetilde{K}}(\pm\lambda_0) = 0$.

**Proof.** Fix $n \geq 5$. Then there exists $M \in C_+^\infty$ such that $\psi^{-1}\rho_M(\lambda)$ is sign-changing

(see [15] Lemma 4.10 where an example of such body is constructed). For $s \in [0,1]$, consider the Minkowski sum $K(s) = sB_2^n + (1-s)M$. Then $\psi^{-1}\rho_{K(0)}(\lambda)$ is sign-

changing and there exists $\Lambda' \subset T^{n-1}$ such that

$\psi^{-1}\rho_{K(0)}(\lambda) < 0, \forall \lambda \in \Lambda'$. On the other hand , $\psi^{-1}\rho_{K(1)}(\lambda) > 0, \forall \lambda \in T^{n-1}$. By the previous lemma the map $s \to \psi^{-1}\rho_{K(s)}(\lambda)$ is continuous, and there is

$s_0 \in [0,1]$ such that

$\psi^{-1}\rho_{K(s_0)}(\lambda) \geq 0, \forall \lambda \in T^{n-1}$ and

$\psi^{-1}\rho_{K(s_0)}(\lambda) = 0, \forall \lambda \in \Lambda \subset T^{n-1}$,

for some $\Lambda \neq \emptyset$. Fix any $\lambda_0 \in \Lambda$. Consider an even $C^\infty$ smooth function $g$ on $T^{n-1}$ such that $g(x) > 0, \forall x \neq \pm\lambda_0$ and $g(\pm\lambda_0) = 0$. For $\epsilon > 0$ define a body $\widetilde{K}$ (depending on $\lambda_0$): $\psi^{-1}\rho_{\widetilde{K}}(\lambda) = \psi^{-1}\rho_{K(s_0)}(\lambda) + \epsilon g(\lambda)$.

Note that $\psi^{-1}\rho_{\widetilde{K}}(\lambda)$ is strictly positive for all

$\lambda \neq \pm\lambda_0$ , and $\psi^{-1}\rho_{\widetilde{K}}(\pm\lambda_0) = 0$. We get

$\rho_{\widetilde{K}}(x) = \rho_{K(s_0)}(x) + \epsilon\psi g(x)$.

Since $\psi g$ is a $C^\infty$ function, and $K(s_0) \in C_+^\infty$, we may choose $\epsilon$ small enough so that $\widetilde{K} \in C_+^\infty$. Using the rotation argument, we can take $\lambda_0$ to be arbitrary.

**Theorem 3.5.** Let $n \geq 5$ be odd. There exists $\epsilon > 0$ and a conver symmetric body $K$ that is not an intersection body, but nevertheless $\forall x \in T^{n-1}$ there exists an intersection body $L_x$ such that $\rho_K = \rho_{L_x}$ on $E_\epsilon(x)$.

**Proof.** We define a convex body $K$ and a family of convex bodies $\{L_x\}_{x\in T^{n-1}}$ using $\widetilde{K}$ and functions $f_{x,\lambda_0}$ from Lemma 3.1. We fix some small $\epsilon$ satisfying the requirements of Lemma 3.1 and we may assume $c = 2\psi^{-1}1$ (see Remark 3.2). Then, define $K = K_{\delta,\lambda_0}$ via $\rho_K = \rho_{\widetilde{K}} - \delta$, where for the moment $\delta > 0$ is assumed to be so small that $K \in C_+^\infty$ and $\psi^{-1}\rho_K$ is strictly positive outside $U_\epsilon(\lambda_0)$. Note that $\psi^{-1}\rho_K(\lambda_0) < 0$ and thus $K$ is not an intersection body.

Now we define a family of convex bodies $\{L_x\}_{x\in T^{n-1}}$. Since $\widetilde{K} \in C_+^\infty$, we take $\delta$ so small that

$\rho_{L_x} := \rho_{\widetilde{K}} - \delta + \delta f_{x,\lambda_0} > 0$ on $T^{n-1}$

and $L_x$ is convex. Observe that

$\rho_{L_x} := \rho_K$ on $E_\epsilon(x)$ for any $x \in T^{n-1}$. We can assume that $\delta$ is so small that

$\psi^{-1}\rho_{L_x} = \psi^{-1}\rho_{\widetilde{K}} - \delta\psi^{-1}1 + \delta\psi^{-1}f_{x,\lambda_0} > 0$

on $\frac{T^{n-1}}{U_\epsilon(\lambda_0)}$, since $\psi^{-1}\rho_{\widetilde{K}} > 0$ on $\frac{T^{n-1}}{U_\epsilon(\lambda_0)}$.

To show that bodies $L_x$ are intersection bodies $\forall x \in T^{n-1}$, it is enough to prove that

$\psi^{-1}\rho_{L_x} > 0$ on $U_\epsilon(\lambda_0)$. By Remark 3.2,

$$\min_{x\in T^{n-1}} \psi^{-1} f_{x,\lambda_0} \geq 2\psi^{-1}1 \quad \text{on} \quad U_\epsilon(\lambda_0),$$

hence

$$\psi^{-1}\rho_{L_x} = \psi^{-1}\rho_{\tilde{K}} - \delta\psi^{-1}1 + \delta\psi^{-1}f_{x,\lambda_0} \geq \delta\psi^{-1}1 > 0$$

on $U_\epsilon(\lambda_0)$. Moreover, $\delta > 0$ can be chosen independently of $x$ since the set of functions $\{f_{x,\lambda}\}_{x,\lambda\in T^{n-1}}$ in Lemma 3.1 is finite.

## 3. There is no local equatorial characterization of of zonoids in odd dimensions.

The proofs in this section are very similar (in fact, almost identical) to the ones in the previous section.

**Lemma 4.1.** Let $n \geq 3$ be odd. Then there exists an $\epsilon > 0$ and an absolute constant $c > 0$ such that for any $x, \lambda \in T^{n-1}$, there exists an even function $f_{x,\lambda}$ satisfying $f_{x,\lambda} = 0$ on $E_\epsilon(x)$, and $Cos^{-1}f_{x,\lambda} \geq c$ on $U_\epsilon(\lambda)$.

**Proof.** The proof follows the same lines as that of Lemma 3.1. One has to change the Spherical Radon transform to the Cosine transform, put support functions instead of radial functions and thus, use section functions of polar bodies together with (2), (4) and (8).

**Remark 4.2.** Note that dilating $M$ and $Q$ (and thus functions $f_{x,\lambda}$) we may assume that $c$ is as large as we want. For technical reasons, we take $c = 2Cos^{-1}1$. Moreover, we can assume that the set of functions $\{f_{x,\lambda}\}_{x,\lambda\in T^{n-1}}$ in the lemma is finite.

**Lemma 4.3.** Let $n \geq 3$. For any point $\lambda_0 \in T^{n-1}$ there exists a zonoid $\widetilde{K} \in C_+^\infty$ such that $Cos^{-1}h_{\widetilde{K}}(\lambda)$ is strictly positive for all

$\lambda \neq \pm\lambda_0$ and $Cos^{-1}h_{\widetilde{K}}(\pm\lambda_0) = 0$.

**Proof.** Fix $n \geq 3$. Then there exists $M \in C_+^\infty$ such that $Cos^{-1}h_M$ is sign-changing

(see [15], the Fourier Analytic solution of Shephard problem for a construction of a $C_+^\infty$ non-zonoid body).

For $s \in [0,1]$ consider the Minkowski sum $K(s) = sB_2^n + (1-s)M$. Then $h_{K(s)} =$ th $B_2^n + (1-s)h_M$ is a $C^\infty$ -function, $Cos^{-1}h_{K(0)}(\lambda)$ is sign-changing and there exists $\Lambda' \subset T^{n-1}$ such that $Cos^{-1}h_{K(0)}(\lambda) < 0, \forall\lambda \in \Lambda'$. On the other hand, $Cos^{-1}h_{K(1)}(\lambda) > 0, \forall\lambda \in T^{n-1}$. The map $s \to Cos^{-1}h_{K(s)}$ is continuous, since $Cos$ is a continuous bijection of $C^\infty(T^{n-1})$ into

itself. (see [6]). Hence, there is $s_0 \in [0,1]$ such that

$Cos^{-1}h_{K(s_0)} \geq 0$, and $Cos^{-1}h_{K(s_0)}(\lambda) = 0$, $\forall \lambda \in T^{n-1}$ and some $\Lambda \neq \emptyset$. Fix any $\lambda_0 \in \Lambda$. Consider an even $C^\infty$ smooth function $g$ on $T^{n-1}$ such that

$g(x) > 0, \forall x \neq \pm\lambda_0$ and $g(\pm\lambda_0) = 0$. For $\epsilon > 0$ define a body $\widetilde{K}$:

$$Cos^{-1}h_{\widetilde{K}}(\lambda) = Cos^{-1}h_{K(s_0)}(\lambda) + \epsilon g(\lambda).$$

Note that $Cos^{-1}h_{\widetilde{K}}(\lambda)$ is strictly positive for all $\lambda \neq \pm\lambda_0$, and

$Cos^{-1}h_{\widetilde{K}}(\pm\lambda_0) = 0$. Moreover, $h_{\widetilde{K}} = h_{K(s_0)} + \epsilon Cosg$.

Since $Cosg$ is a continuous function and $K(s_0) \in C_+^\infty$, we may choose $\epsilon$ small enough so that $\widetilde{K} \in C_+^\infty$. Using the rotation argument, we can take $\lambda_0$ to be arbitrary.

**Theorem 4.4.** Let $n \geq 3$ be odd.There exists $\epsilon > 0$ and a convex body $K$ that is not a zonoid, but nevertheless $\forall x \in T^{n-1}$ there exists a zonoid $L_x$ such

$$h_K = h_{L_x} \text{ on } E_\epsilon(x).$$

**Proof.** We define a convex body $K$ and a family of convex bodies $\{L_x\}_{x\in T^{n-1}}$ using the zonoid $\widetilde{K}$ and functions $f_{x,\lambda_0}$ from Lemma 4.1. We fix some small $\epsilon$ satisfying the requirements of Lemma 4.1 with $c = 2Cos^{-1}1$ (see Remark 4.2). Then, define $K = K_{\delta,\lambda_0}$ via $h_K = h_{\widetilde{K}} - \delta$, where for the moment $\delta > 0$ is assumed to be so small that $K \in C_+^\infty$ and $Cos^{-1}h_K$ is strictly positive outside $U_\epsilon(\lambda_0) < 0$ and thus $K$ is not a zonoid. Now we define a family of convex bodies $\{L_x\}_{x\in T^{n-1}}$. Since $\widetilde{K} \in C_+^\infty$, we take $\delta$ so small that $h_{L_x} := h_{\widetilde{K}} - \delta + \delta f_{x,\lambda_0} > 0$ on $T^{n-1}$ and $L_x$ is convex. Observe that $h_{L_x} = h_K$on $E_\epsilon(x)$ for any $x \in T^{n-1}$. We can assume that $\delta$ is so small that

$Cos^{-1}h_{L_x} = Cos^{-1}h_{\widetilde{K}} - \delta Cos^{-1}1 + \delta Cos^{-1}f_{x,\lambda_0} > 0$ on $\frac{T^{n-1}}{U_\epsilon(\lambda_0)}$, since $Cos^{-1}h_{\widetilde{K}} > 0$ on $\frac{T^{n-1}}{U_\epsilon(\lambda_0)}$.

To show that bodies $L_x$ are zonoids $\forall x \in T^{n-1}$, it is enough to prove that $Cos^{-1}h_{L_x} > 0$ on $U_\epsilon(\lambda_0)$. By Remark 4.2, $\min_{x\in T^{n-1}} Cos^{-1}f_{x,\lambda_0} > 2Cos^{-1}1$ on $U_\epsilon(\lambda_0)$, hence $Cos^{-1}h_{L_x} = Cos^{-1}h_{\widetilde{K}} - \delta Cos^{-1}1 + \delta Cos^{-1}f_{x,\lambda_0} \geq \delta Cos^{-1}1 > 0$

on $U_\epsilon(\lambda_0)$, and the result follows.

## 4. There is a local equatorial characterization of intersection bodies and zonoids in even dimensions.

We consider at first intersection bodies. The proof of the following lemma is obtained by a straightforward repetition of the argument from (see [15]), and we omit the details.

**Lemma 5.1.** Let $g(x)$ be an even homogeneous function of degree-1 such that $g(x)$ is nonnegative and infinitely smooth on $T^{n-1}$. Then

$$\tilde{g}(\lambda) = (-1)^{\frac{n-2}{2}} \pi A_{g,\lambda}^{n-2}(0),$$

where

$$A_{g,\lambda}(z) = \int_{\{y \in \mathbb{R}^n : y.\lambda = z\}} \chi_{[0,1]}\left(\frac{1}{g(y)}\right) dy, \lambda \in T^{n-1}.$$

**Theorem 5.2.** Let $n$ be even and let $K \subset \mathbb{R}^n$ be an origin symmetric convex body. Assume that for any great sphere $\lambda^{\perp} \cap T^{n-1}$, there exists an intersection body $L_\lambda$ and a neighborhood $E_{\epsilon(\lambda)}(\lambda)$ of $\lambda^{\perp} \cap T^{n-1}$ such that the radial functions of $K$ and $L_\lambda$ coincide at all points of $E_{\epsilon(\lambda)}(\lambda)$; then $K$ is an intersection body

**Proof.** If $K$ and $L_\lambda$ are infinitely smooth, then it is enough to observe that

$$\rho_K(u) = \rho_{L_\lambda}(u) \; \forall u \in E_{\epsilon(\lambda)}(\lambda)$$

implies $A_{K,\lambda}(s) = A_{L_\lambda,\lambda}(s)$ for sufficiently small $s$ and apply (5).

Consider the general case case. It was proved by A. Koldobsky that an original-symmetric convex body $K$ is an intersection body if and only if $\rho_K$ represents a positive definite distribution (see for example, Theorem 4.1 in [15]). Thus, it is enough to show that $\langle \widehat{\rho_K}, \eta \rangle \geq 0$, for all nonnegative test functions $\eta$ on $\mathbb{R}^n$.

Using the definition of the Fourier Transform of distributions (see section 2.5, in [15] ) and passing to the polar coordinates, we get

$$\langle \widehat{\rho_K}, \eta \rangle = \langle \rho_K, \hat{\eta} \rangle = \int_{\mathbb{R}^n} \rho_K(x)\, \hat{\eta}(x) dx$$

$$= \int_{T^{n-1}} \rho_K(\theta) \int_0^{\infty} r^{n-2} \hat{\eta}(r\theta) dr d\theta.$$

Observe that the function

$$\alpha(x) := \int_0^{\infty} r^{n-2} \hat{\eta}(rx) dr, x \in \frac{R^{n-1}}{\{0\}}$$

is homogeneous of degree-$n+1$ and infinitely smooth. Hence, we may apply equality 4.3, page 72 together with Lemma 3.7, page 53 from ([15]) to claim that there exists an infinitely smooth non-negative homogeneous of degree $-1$ function

$g(x) = \frac{1}{2} \int_{\mathbb{R}} \eta(sx)\, ds$, such that $\hat{g}(\theta) = \alpha(\theta) \; \forall \theta \in T^{n-1}$.

Thus,

$\int_{T^{n-1}} \rho_K(\theta) \int_0^\infty r^{n-2} \hat{\eta}(r\theta) dr d\theta = \int_{T^{n-1}} \rho_K(\theta)\, \hat{\eta}(\theta) d\theta$.

Using a partition of unity on $T^{n-1}$ together with homogeneity of $g$, we can write

$$g(\theta) = \sum_{j=1}^{m} g_j(\theta) = \sum_{j=1}^{m} \frac{1}{2} \int_{\mathbb{R}} \eta_j(s\theta)\, ds, \theta \in T^{n-1},$$

where $\operatorname{supp} g_j|_{T^{n-1}} \subset U_{\epsilon_j}(\lambda_j)$ are small enough.

By the previous lemma, $\operatorname{supp} g_j|_{T^{n-1}} \subset U_{\epsilon_j}(\lambda_j)$ implies $\operatorname{supp} \hat{g}_j|_{T^{n-1}} \subset U_{\epsilon_j}(\lambda_j)$.

Hence,

$$\langle \widehat{\rho_K}, \eta \rangle = \sum_{j=1}^{m} \int_{T^{n-1}} \rho_K(\theta)\, \hat{g}_j(\theta) d\theta$$

$$= \sum_{j=1}^{m} \int_{E_{\epsilon_j}(\lambda_j)} \rho_K(\theta)\, \hat{g}_j(\theta) d\theta$$

$$= \sum_{j=1}^{m} \int_{E_{\epsilon_j}(\lambda_j)} \rho_{L_{\epsilon_j}}(\theta)\, \hat{g}_j(\theta) d\theta$$

$$= \sum_{j=1}^{m} \int_{T^{n-1}} \rho_{L_{\epsilon_j}}(\theta)\, \hat{g}_j(\theta) d\theta$$

$$= \sum_{j=1}^{m} \langle \widehat{\rho_{L_{\epsilon_j}}}, \eta_j \rangle \geq 0.$$

The following result was obtained independently by [23] and[11]. Its proof could be also obtained by the arguments similar to those in the previous proof, and we omit it.

**Theorem 5.3.** Let $n$ be even and let $K \subset \mathbb{R}^n$ be an origin symmetric convex body. Assume that for any great sphere $\lambda^\perp \cap T^{n-1}$, there exists a zonoid $Z_\lambda$ and a neighborhood $E_{\epsilon(\lambda)}(\lambda)$ of $\lambda^\perp \cap T^{n-1}$ such that the boundaries of $K$ and $Z_\lambda$ coincide at all points where the exterior unit vector belong to $E_{\epsilon(\lambda)}(\lambda)$; then $K$ is a zonoid.

## 5. There is no local equatorial characterization of intersection bodies .

In this section we prove the analog of the result of [28] for zonoids. Our proof is different from the one of W. Weil. We show that, given $x, \lambda \in T^{n-1}$, one can construct a function $f$ which is zero around $x$, but such that the inverse spherical Radon transform of $f$ is positive around $\lambda$. For convenience of the reader we split the proof of this auxiliary result (see Lemma 6.4) into four statements. We will use the following notation

$$\Omega_{\epsilon,x} = \{f \in C^\infty(T^{n-1}) : f = 0 \text{ on } U_\epsilon(x)\}, \quad 0 < \epsilon < 1.$$

**Lemma 6.1.** Let $n \geq 3$, and let $\lambda, x \in T^{n-1}$ be two orthogonal vectors. Assume that any $f \in \Omega_{\frac{1}{4},x}$ satisfies $\psi^{-1}(\lambda) = 0$. Then for any pair of orthogonal vectors

$u, v \in T^{n-1}$ we have $f \in \Omega_{\frac{1}{4},u}$ implies $\psi^{-1}f(v) = 0$.

**Proof.** For any two pairs of orthogonal unit vectors $(\lambda, x), (u, v)$ there exists a rotation $\rho \in TO(n)$ satisfying $u = \rho(x), v = \rho(\lambda)$. Since $\psi^{-1}$ commutes with rotation, the result follows.

**Lemma 6.2.** Let $n \geq 3$, and let $\lambda \in x^{\perp}$. Assume that any $f \in \Omega_{\frac{1}{4},x}$ satisfies $\psi^{-1}f(\lambda) = 0$. Then $\psi^{-1}\left(\Omega_{\frac{1}{2},x}\right) \subset \Omega_{\frac{1}{4},\lambda}$

**Proof.** Take any $u \in U_{\frac{1}{4}}(\lambda)$.

Let $\rho \in TO(n),\ \rho(\lambda) = u,$

where $\lambda$ is rotated into $u$ inside $U_{\frac{1}{4}}(\lambda)$ in the plane containing $\lambda, u$ and the origin. Then $\rho(x) \in U_{\frac{1}{4}}(x)$, and

$\Omega_{\frac{1}{2},x} \subset \Omega_{\frac{1}{4},\rho(x)}$. Moreover, $\psi^{-1}f(u) = 0$ since $\psi^{-1}$ commutes with rotations. The point $u$ was chosen arbitrarily in $U_{\frac{1}{4}}(\lambda)$, hence $\psi^{-1}\left(\Omega_{\frac{1}{2},x}\right) \subset \Omega_{\frac{1}{4},\lambda}$ .

**Lemma 6.3.** Let $n \geq 3$, and let $\lambda \in x^{\perp}$. Then there exists a function $f = f_{x,\lambda}$ on $T^{n-1}$ satisfying $f_{x,\lambda} = 0$ on $U_{\frac{1}{4}}(x)$, but $\psi^{-1}f_{x,\lambda}(\lambda) \neq 0$.

**Proof.** Assume the contrary. Then $\psi^{-1}\left(\Omega_{\frac{1}{2},x}\right) \subset \Omega_{\frac{1}{4},\lambda}$ by Lemma 6.2. Take any vector $y \in T^{n-1}$, and find a vector $q \in x^{\perp} \cap y^{\perp}$. Let $\rho \in TO(n)$ by such that $\rho(x) = x, \rho(\lambda) = q$. Observe that $f \in \Omega_{\epsilon,x}$ implies $f(\rho(.)) \in \Omega_{\epsilon,x}$. Since $\psi^{-1}$ commutes with rotations, $\psi^{-1}\left(\Omega_{\frac{1}{2},x}\right) \subset \Omega_{\frac{1}{4},\lambda}$ yields $\psi^{-1}\left(\Omega_{\frac{1}{2},x}\right) \subset \Omega_{\frac{1}{4},q}$. Take two pairs of orthogonal vectors $(x, q)$ and $(q, y)$. By Lemma 6.1, we have

$\psi^{-2}f(y) = 0$. Thus $\psi^{-2}f \equiv 0$, a contradiction.

**Lemma 6.4.** Let $n \geq 3$. Then there exists an $\epsilon > 0$ and an absolute constant $c > 0$ such that for any $x, \lambda \in T^{n-1}$, there exists an even functional $f_{x,\lambda}$ satisfying $f_{x,\lambda} = 0$ on $U_{\epsilon}(x)$, and $\psi^{-1}f_{x,\lambda} \geq c$ on $U_{\epsilon}(\lambda)$.

**Proof.** We fix points $x$ and $\lambda$, and provide an $\epsilon > 0$ and $c > 0$ depending on $x, \lambda$ such that there is a function $f_{x,\lambda}$ satisfying $f_{x,\lambda} = 0$ on $U_{\epsilon}(x)$, and $\psi^{-1}f_{x,\lambda} \geq c > 0$ on $U_{\epsilon}(\lambda)$. Then we use the compactness argument to prove the statement of the lemma. Let $\lambda \notin x^{\perp}$. Then there exists an $\epsilon > 0$, such that $\lambda \notin E_{\epsilon}(x)$. For any function $g$ the values of $\psi g$ on $U_{\epsilon}(x)$ depend only on the values of $g$ on $E_{\epsilon}(x)$. Hence, we may consider an even

$C^\infty$ −function $g$ such that $g(\pm\lambda) > 0$ and $g(v) = 0$, for $v \in E_\epsilon(x)$ and define $f_{x,\lambda} = \psi g(x)$.

Let $\lambda \in x^\perp$. Then, the previous lemma implies the existence of $= \epsilon(x,\lambda) = \frac{1}{8}$,

and a function $f = f_{x,\lambda}$ on $T^{n-1}$ satisfying $f_{x,\lambda} = 0$ on $U_\epsilon(x)$, but $\psi^{-1} f_{x,\lambda}(\lambda) > 0$

(change the sign of $f_{x,\lambda}$ if necessary).

Thus, we proved that for any $x$ and $\lambda$, there is $\epsilon' = \epsilon'(x,\lambda) > 0$

and there is a function $f_{x,\lambda}$ such that $f_{x,\lambda} = 0$ on $U_{\epsilon'}(x)$, but

$$\psi^{-1} f_{x,\lambda}(\pm\lambda) \geq c',\ c' = c'(x,\lambda) > 0.$$

From the continuity of the function $\psi^{-1} f_{x,\lambda}$ we get that

$$\psi^{-1} f_{x,\lambda} \geq c,\ c = c(x,\lambda) > 0$$

on $U_{\epsilon''}(\lambda)$, for some $\epsilon'' > 0$.

Take $\tilde{\epsilon} = \tilde{\epsilon}(x,\lambda) = \min(\epsilon', \epsilon'')$.

We show that for any $x$ and $\lambda$, there is

$\tilde{\epsilon} = \tilde{\epsilon}(x,\lambda) > 0$ and there is a function $f_{x,\lambda}$ such that $f_{x,\lambda} = 0$ on $U_{\tilde{\epsilon}}(x)$, but $\psi^{-1} f_{x,\lambda} \geq c$ on $U_{\tilde{\epsilon}}(\lambda), c = c(x,\lambda) > 0$.

Now we use the compactness argument to prove that we can choose an $\epsilon$ and $c$ independent of $x$ and $\lambda$. We choose a finite set of $\{x_i, \lambda_i\}_{i=1}^m$ such that

$\left\{U_{\frac{\tilde{\epsilon}_i}{2}}(x_i) \times U_{\frac{\tilde{\epsilon}_i}{2}}(\lambda_i)\right\}_{i=1}^m$ covers $T^{n-1} \times T^{n-1}$. We take

$$\epsilon = \frac{1}{2} \min_{1\leq i\leq m} \tilde{\epsilon}_i \text{ and } c = \min_{1\leq i\leq m} c(x_i, \lambda_i).$$

Then for any $(x,\lambda)$ there is a $(x_i,\lambda_i)$ such that

$$U_\epsilon(x) \times U_\epsilon(\lambda) \subset U_{\tilde{\epsilon}_i}(x_i) \times U_{\tilde{\epsilon}_i}(\lambda_i)$$

and we may define $f_{x,\lambda} = f_{x_i,\lambda_i}$ .

**Theorem 6.5.** Let $n \geq 5$. There exists a conver body $K$ that is not an intersection body, such that $\forall \in xT^{n-1}$ there exists an $\epsilon(x)$ and an intersection body $L_x$ such that $\rho_K = \rho_{L_x}$ on $U_{\epsilon(x)}(x)$.

**Proof.** Repeat the Proof of Theorem 1(see [22]).

## Conclusion

Finally we have show that there is no local equatorial characterization of zonoids in odd dimensions. This gives a negative answer to the conjecture posed by W.Weil in 1977 and shows that the local equatorial characterization of zonoids may be given

only in even dimensions. In addition we prove a similar result for intersection bodies and show that there is no local characterization of these bodies.

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