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The Libyan Journal of Science

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Prevalence of Mutations in TAL1 Gene in Individuals With T-ALL and T-NHL

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Abstract

Mutations in the TAL1 (T-cell acute leukemia 1) gene were recently described in patients with T-cell acute lymphoblastic leukaemia (T-ALL) and in those with lymphoblastic T-cell non-Hodgkin’s lymphoma (T-NHL). The purpose of this pilot study was to assess the prevalence of mutations in TAL1 gene in T-ALL and T-NHL. DNA samples from 15 unrelated healthy controls, 20 T-ALL patients, and 10 T-NHL patients were analyzed using DNA-PCR and direct DNA sequencing to identify sequence genetic variations in TAL1 gene (exons 2 and 3). TAL1 exon 2 mutations were identified in 7.7% adult and 12.5% adolescent T-ALL patients analyzed. TAL1 exon 2 mutations were detected in 16.7% of the adult T-NHL patients analyzed. Sequencing of TAL1 exon 3 showed no sequence variation for the T-ALL and T-NHL cancer patients analyzed. No sex difference where observed in the incidences of TAL1 exons 2 mutations between T-ALL and T-NHL patients with and without TAL1 mutations. TAL1 exon 2 missense and frame-shift mutations were present in 44.4% (4/9) and 55.6% (5/9) of T-ALL patients, respectively. However, the frame-shift and missense mutations in the T-NHL patients accounted for, where respectively, 60% (3/5) and 40% (4/5) of all TAL1 exon 2 mutations.

Comparing the clinical features showed that there are no differences in PLT and WBC counts as well as the average age between T-ALL and T-NHL patients with and without TAL1 mutations. Overall, these findings indicate that TAL1 mutations are too rare to be of clinical relevance, and do not seem to be significantly associated with the increased T-ALL and T-NHL susceptibility, implying different pathways with respect to TAL1 genetic polymorphisms as a risk factor for T-ALL and T-NHL at least in this population of Libyans.

Keywords: T-cell acute lymphoblastic leukaemia (T-ALL); T-cell non-Hodgkin’s lymphoma (T-NHL); T-cell acute leukemia 1 (TAL1) gene; mutation; susceptibility.

Accepted for publication: 8/12/2016
المستخلص

أثبتت الدراسات الحديثة أن وجود طفرات في مورثة TAL1 مرتبطة بمرضى سرطان T-ALL. هدفت هذه الدراسة إلى تقييم مدى انتشار الطفرات في مورثة TAL1 في مرضى T-NHL و T-ALL. تم تجميع عدٍّ 15 عينة من الخلايا النموية من أشخاص أصحاء، و 20 عينة من مرضى T-ALL و 10 عينات من مرضى T-NHL وتحليل المادة الوراثية باستخدام تكنولوجيا DNA Sequencing و DNA-PCR. وذلك لتحديد التغيرات الوراثية في تتابعات الأكسون 2 و3 من مورثة TAL1.

أوضح نتائج هذه الدراسة وجود طفرات في الأكسون 2 في 7.7% من البالغين و12.5% من المراهقين من مرضى T-ALL. أظهرت النتائج أيضا وجود طفرات في الأكسون 2 في حوالي 16.7% من البالغين المصابين بمرض T-NHL. لم تظهر نتائج هذه الدراسة وجود طفرات في الأكسون 3 من نفس المورثة في كلاً من مرضى T-ALL و T-NHL. كما لم تظهر النتائج وجود فروق معنوية بين الجنسين من حيث وجود طفرات في الأكسون 2 للمورثة TAL1 بين مرضى T-ALL ومرضى T-NHL. الطفرات المطلوبة وطفرات الإزاحة في الأكسون 2 للمورثة TAL1 كانت تصل 44.4% و55.6% على التوالي في مرضى T-ALL. شكلت الطفرات المطلوبة وطفرات الإزاحة في مرضى T-ALL حوالي 60% و 40% على التوالي من الطفرات في الأكسون 2. أظهرت النتائج السريرية عدم وجود اختلاف معنوي في عدد كلاً من الصفائح الدموية و كرات الدم البيضاء، فضلاً عن مستوى العمر بين مرضى T-ALL ومرضى T-NHL سواء عند أولئك الذين ليس لديهم أو لديهم طفرات في الأكسون 2 للمورثة TAL1.

تشير نتائج هذه الدراسة إلى أن الاختلافات في مورثة TAL1 ليست ذات أهمية طبية، ولا يبدو أنها تترافق مع زيادة القابلية للإصابة بمرض T-ALL ومرض T-NHL، مما يدل على وجود مسارات أخرى تتعلق بخطر الإصابة بمرض T-ALL ومرض T-NHL على الأقل بالنسبة لهذه العينة من المرضى البحرينيين.

Introduction

T-cell acute lymphoblastic leukemia (T-ALL) and lymphoblastic T-cell non-Hodgkin’s lymphoma (T-NHL) are closely related disorders and although T-ALL cells are more often similar to the early stages of thymocytes than T-NHL cells, there is a considerable overlap between these two disorders [1-2]. Morphology, ultrastructure and immunocytochemistry do not allow us to distinguish them and even clinically, a clear cut discrimination between T-ALL and T-NHL is not evident [3]. In fact, T-NHL is usually differentiated from T-ALL by minimal or absence of bone marrow and peripheral blood involvement, normal white blood
Prevalence of Mutations in TAL1 Gene in Individuals with T-ALL and T-NHL

cell (WBC) count, normal hemoglobin levels, and lack of organomegaly. Because
this distinction does not apply in every case, a presence of less than 25% of blasts
in the bone marrow is currently used to define T-NHL from T-ALL [4]. T-ALL
occurs by the uncontrolled proliferation of T-lymphoid precursors arrested during
distinct stages of differentiation [5]. The percentage of T-ALL in India is very
high (43.1%) when compared to the Western countries (15-25%) [6]. TAL-1
deletion is the most common genetic abnormality in T-ALL [5].

The T-Cell Acute Lymphocytic Leukemia 1 (TAL1) gene (also known as SCL
or TCL5) located on 1p32, encodes a basic helix loop helix protein TAL-1 [7] and
is essential for the earliest stages of hematopoietic stem cell development and
differentiation [7]. Translocation of TAL1 (1p32) next to the TCRd loci in the
t(1;14) (p32;q11), occurs in 3% of T-cell ALL and results in aberrant TAL1
expression [5]. A frequent mode of TAL1 deregulation is a site-specific deletion
(TAL1 deletion) of ~ 90 kb. As a result the coding exons of the TAL1 gene are
juxtaposed to the first non-coding exon of the SIL gene, which is almost
completely deleted [8]. The expressed SIL-TAL1 fusion transcript produces a
normal TAL1 protein, but it is transcriptionally controlled by the SIL gene
promoter [9]. Cytogenetic investigations of large NHL series reported abnormal
cytotypes, often with complex abnormalities, in about 85% of tumors.
Chromosome 1p aberrations both as structural and numerical abnormalities were
found to be one of the most frequently occurring aberrations among T-cell
neoplasias. The most frequent 1p breakpoints involve band 1p32-36 [10].
Interestingly, both deletions and translocations frequently observed in T-ALL
involve this same chromosomal region [11], the most common genetic change
observed being a 90 Kb deletion resulting in fusion of the SIL promoter to the
TAL1 gene [8, 12-15]. TAL1 deletions are not detected by classical cytogenetics.
SIL contains three donor deletion sites (Sildb1 to Sildb3), of which Sildb1 is the
most commonly used (98% of cases). TAL1 contains seven acceptor deletion sites
(taldb1 to taldb7), with two being involved in almost all cases (taldb1 and taldb2).
The vast majority (~90%) of TAL1 deletions is located between Sildb1 and taldb1,
and is known as TAL1 deletion type I. The TAL1 deletion type-2 occurs between
Sildb1 and taldb2 [15]. Disruption of the TAL1 locus on chromosome 1p32
represents one of the most commonly recognized genetic abnormalities in T-ALL.
TAL1 codes for a basic helix-loop-helix (bHLH) transcription factor [7, 16] which
is normally expressed by haemopoietic precursors, by the megakaryocyte,
mastocyte and erythroid lineages and by endothelial cells [17] but not by normal
T cells. The TAL1 gene is disrupted by at least two mechanisms: translocation and
site-specific deletion [16]. The t(1;14) (p32;q11), involving TAL1 and the TCRd
locus on chromosome 14q11, occurs in 3% of childhood T-ALL [7, 9, 12] and
rare variant translocations [9] have been identified. A more frequent,
approximately 90 kbp, site-specific deletion upstream to TAL1, tald, leads to replacement of the 50 part of TAL1 by regulatory sequences from the SIL gene. It occurs in 6–26% of T-ALL, preferentially in cases which express TCR ab [18-19]. At least five TAL1 deletion breakpoints have been reported [19], with taldb1 and, to a lesser extent, taldb2 occurring most frequently. Detection of tald is most frequently undertaken by PCR from DNA, with screening limited to detection of tald1 and tald2. Several primer pairs or Southern blot analysis with SIL and 50 TAL1 probes are necessary to detect all variant breakpoints [20].

However, to the best of our knowledge, the role of TAL1 genetic polymorphisms in the susceptibility, clinical features and biological characteristics of T-ALL and T-NHL remain unknown at least in Libya. Therefore, this study aims, for the first time, to explore the possible association between the TAL1 genetic variations (exons 2 and 3) and T-ALL and T-NHL risk using DNA-PCR and direct DNA sequencing. Also, clarifying the effect of TAL1 mutations on clinical parameters is of particular interest in our study.

Materials and Methods

Patient Population

The case–control study consisted of 15 controls, 20 T-ALL patients and 10 T-NHL patients. Unrelated healthy controls were recruited from general population. T-ALL and T-NHL patients attending to Department of Oncology, Triploli Medical Cancer, at Tripoli, Libya were included in the present study. For sample preservation and genetic analysis, local institutional review board approval and informed consent from all participants were obtained by the Ethics Committee of the First Affiliated Tripoli Medical Cancer at Tripoli, Libya. T-ALL and T-NHL cancer was confirmed by histological examination after hysterectomy or myomectomy. The median age of the case series was 23 years (range, 3 - 66 years), and all of studied groups were males (60.5%), and all were Libyans (undefined ethnic group).

Sampling and DNA Extraction

Blood samples (five-mL) were taken from all participants by peripheral antecubital venous puncture, drawn into sodium EDTA tube and were then stored at −20°C until analysis. Genomic DNA was extracted from the blood samples of controls, T-ALL patients, and T-NHL patients by a standard procedure (standard phenol and chloroform) [20]. The integrity of extracted genomic DNA and its concentration were measured by UV-spectrophotometry (BioPhotometer, eppendorf), with absorbance of A260/A280 nm ratios at pH 8.0 between 1.7 and 2
for all samples, and ethidium-bromide fluorescence of DNA separated by 1.5% AGE (agarose gel electrophoresis). The quality was checked by amplifying β-actin housekeeping gene. Purified DNA was stored at -70 °C till being used.

**Molecular Characterization of T-ALL and T-NHL Patient Samples**

*TAL1* mutations were analyzed by PCR amplification of *TAL1* exons 2 and 3, and followed by direct bidirectional DNA sequencing using previously published primers and PCR conditions [5, 21].

PCR amplification was carried out using a Perkin-Elmer 480 thermocycler (Applied Biosystems) in a 25 µl reaction mixture containing 10X PCR buffer, 1.5 mM MgCl₂, 200 µM of dNTP’s (AB Gene), 1 U of Hotstart Taq Polymerase (AB Gene), 10 pmol of forward and reverse primers with 100 ng of genomic DNA (Table 1). The reaction was heated at 95°C for 5 min, then amplified for 30 cycles [95°C/30 sec, annealing/30 sec (57°C for exon 2 and 50°C for exon 3) and 72°C/30 sec] followed by 5 min final extension at 72°C. PCR amplification and the absence of primer dimers were confirmed by analysis of cycling and melting curves. Negative controls (water instead of DNA) were included in all PCR experiments. Linearity and specificity of PCR amplification were validated before quantification. *TAL1* exons 2 and 3 amplified products (198 bp and 235 bp, respectively) were visualized by electrophoresing on a 1.5% AGE. All PCR amplifications resulted in a single and specific product of the expected size (Figure 1). The banding pattern of extracted genomic DNA did not show any evidence of DNA degradation (Figure 1).

Qualified amplicons (PCR products) were cleaned using exonuclease I and shrimp alkaline phosphatase (SAP). Sequencing reaction was performed using Big Dye Terminator Cycle Sequencing Ready Reaction kit v3.0 (Applied Biosystems) at 5’→3’ direction, as indicated by the manufacturer. The nucleotide sequence detection was performed in the ABI Prism 310 Genetic Analyzer (Applied Biosystems) using standard protocols. Finally, the sequencing results were compared with the standard sequence of *TAL1* gene (exons 2 and 3).

**Statistics**

All calculations were performed using the SPSS software package (version 20.0). Data were expressed as mean ± SEM. The patients’ characteristics were analyzed by the chi-squared (χ²) or Fisher’s exact tests for univariate analysis. Potential confounders, such as age, sex, and biochemical measurements were also studied. Differences between controls and cases were compared by either Student’s t-test or one way ANOVA, as appropriate. *P* values less than 0.05 were deemed statistically significant.
Table 1. Primers used to amplify exons 2 and 3 of the TAL1 gene.

<table>
<thead>
<tr>
<th>Exon</th>
<th>Primer sequence</th>
<th>Amplicon size (bp)</th>
<th>Annealing temperature</th>
</tr>
</thead>
</table>
| 2    | FP: 5’GGATTGTGAGAGTGCGTTCA3’  
      | RP: 5’TCAGATCCCTGCTGAGAACA3’ | 198                 | 57 °C                 |
| 3    | FP: 5’ATGTATTCGGGAGCCAGTTG3’  
      | RP: 5’ACCCACGAAACTGACCAGAA3’ | 235                 | 50 °C                 |

FP: forward primer; RP: reverse primer; bp: base pair; TAL1 gene: T-Cell Acute Lymphocytic Leukemia 1 gene.

Reproducibility of the results was confirmed by randomly repeating the mutation analysis for 5% of all DNA samples. The sequencing was carried out in the Biochemistry Department, Hospital of Infants, Tunisia.

Figure 1. Gel electrophoresis of TAL1 exons 2 and 3 PCR products using allele-specific primers. (A) Lane 1 represents the negative control; lanes 2 to 6 show amplified products of TAL1 exon 2 (198 bp); lane 7 correspond to the 500 bp DNA marker. (B) Lane 1 corresponds to the negative control; lanes 2 to 5 show amplified products of TAL1 exon 3 (235 bp); lane 6 corresponds to the molecular size marker 500 bp. TAL1 gene: T-Cell Acute Lymphocytic Leukemia 1 gene; PCR: polymerase chain reaction; bp: base pair.

Results

**TAL1 Gene Mutations and T-ALL Patient Samples**

TAL1 exon 2 mutations were identified in 7.7% adult and 12.5% adolescent T-ALL patients analyzed. Overall, nine TAL1 mutations were documented in this study, involving exon 2. Among these, frame-shift and missense mutations accounted for 44.4% (4/9) and 55.6% (5/9), respectively, of all TAL1 exon 2 mutations. These mutations have not been described previously. The frequency of
Prevalence of Mutations in TAL1 Gene in Individuals with T-ALL and T-NHL

Specific TAL1 mutations identified in the present study is listed in Table 2 and graphically depicted in Figure 2. Furthermore, in our study sequence TAL1 exon 3 revealed no nucleotide variation for the T-ALL cancer patients analyzed, suggesting that TAL1 exon 3 is not, to date, the main known genetic risk factor in T-ALL at least in this study.

Table 2. Characteristics of T-All patients with a TAL1 gene mutation.

<table>
<thead>
<tr>
<th>Case n. (ALL)</th>
<th>Sex</th>
<th>Age</th>
<th>Mutation(s)</th>
<th>Exon</th>
<th>Mutation Type</th>
<th>Protein level</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (ALL) M 15</td>
<td></td>
<td></td>
<td>1022insC</td>
<td>2</td>
<td>Frame-shift</td>
<td>Val340fsX</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1024T&gt;A</td>
<td></td>
<td>Missense</td>
<td>Val341Asp</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1060T&gt;G</td>
<td></td>
<td>Missense</td>
<td>Val353Gly</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1066delIC</td>
<td></td>
<td>Frame-shift</td>
<td>Ala355fsX</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1144T&gt;G</td>
<td></td>
<td>Missense</td>
<td>Val381Gly</td>
<td>N</td>
</tr>
<tr>
<td>2 (ALL) F 35</td>
<td></td>
<td></td>
<td>991delA</td>
<td>2</td>
<td>Frame-shift</td>
<td>Asp331fsX</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1036delIC</td>
<td></td>
<td>Frame-shift</td>
<td>Thr345fsX</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1086C&gt;A</td>
<td></td>
<td>Missense</td>
<td>His360Gln</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1087A&gt;T</td>
<td></td>
<td>Missense</td>
<td>Thr361Ser</td>
<td>N</td>
</tr>
</tbody>
</table>

M: male; F: female; T-ALL: T-cell acute lymphoblastic leukaemia; TAL1 gene: T-cell acute leukemia 1 gene; ins: insertion; del: deletion; R: reported; N: novel; X: Stop codon; fs: frame shift.

TAL1 Mutations and T-NHL Patient Samples

TAL1 exon 2 mutations were detected in 16.7% of the adult T-NHL patients analyzed. Overall, 5 TAL1 mutations were documented in this study, involving exons 2. Among these, missense and frame-shift mutations accounted for, respectively, 60% (3/5) and 40% (4/5) of all TAL1 exon 2 mutations. The frequency of specific TAL1 exon 2 mutations identified in the present study listed in Table 3 and graphically depicted in Figure 3. Sequence analysis of TAL1 exon 3 revealed no nucleotide changes for the T-NHL cancer patients analyzed.

Taken together, our results illustrate that a variation in TAL1 exon 2, but not exon 3, gene play a role in the T-ALL and T-NHL in the studied population.

Association of TAL1 Gene Mutations with T-All and T-NHL Patient Clinical Features

The clinical features of TAL1 wild type and TAL1 mutant T-All and T-NHL cases are compared in Table 4. T-All and T-NHL cases with TAL1 exon 2 positive mutations had a higher PLT counts (P=0.177 and P=0.165, respectively). T-All cases, but not T-NHL cases, with TAL1 exon 2 positive mutations had a lower WBC than the cases with TAL1 exon 2 negative mutations counts (Table 4). The average age of the T-All cases with TAL1 exon 2 mutations was higher than the average age of the cases without TAL1 exon 2 mutations, but they have not
Figure 2. TAL1 gene mutations in T-ALL patients. (A-C) Representative DNA sequencing chromatograms of T-ALL genomic DNA samples showing mutations in exon 2 of TAL1. WT: wild type. T-ALL: T-cell acute lymphoblastic leukaemia; TAL1 gene: T-cell acute lymphoblastic leukemia 1 gene.

reached statistical significance ($P=0.588$). In contrast, the average age of the T-NHL cases with TAL1 exon 2 mutations was lower than the average age of the cases without TAL1 exon 2 mutations ($P=0.576$).

Discussion

T-cell acute lymphoblastic leukemia (T-ALL) and lymphoblastic T-cell non-Hodgkin's lymphoma (T-NHL) are closely associated disorders, and distinguishing between the two is difficult [1-2, 22-24]. TAL1 is amongst the most frequently deregulated oncogenes [22]. In physiological conditions, TAL1 is a regulatory gene that promotes access to alternative fates in hematopoiesis. TAL1 gene alterations and T-ALL and T-NHL susceptibility has been investigated in many studies [22-25], but the reported results are not always consistent. Thus, the present study was designed to identify the prevalence of TAL1 gene (exons 2 and 3) alterations in T-ALL and T-NHL Libyan patients.

TAL1 gene (exons 2) mutations accounts for 15% of pediatric and 25% of adult T-ALL cases [26]. Importantly, significant differences in outcome are present between paediatric and adult T-ALL [26]. In spite of > 70% of children achieve long lasting complete remissions, only 50% of adult T-ALL patients are currently
Prevalence of Mutations in TAL1 Gene in Individuals with T-ALL and T-NHL
cured. In addition, pediatric and adult T-ALLs exhibit marked differences in the
frequency of specific genetic lesions [27-29]. For example, chromosomal
translocation and aberrant expression of the TAL1 and TLX3 oncogenes are highly
prevalent in children, but rare in adults. In contrast, translocations activating TLX1

Table 3. Characteristics of T-NHL patients with a TAL1 gene mutation.

<table>
<thead>
<tr>
<th>Case n.</th>
<th>Sex</th>
<th>Age</th>
<th>Mutation(s)</th>
<th>Exon</th>
<th>Mutation Type</th>
<th>Protein level</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (NHL)</td>
<td>M</td>
<td>3</td>
<td>1098delAG</td>
<td>1056delG</td>
<td>1014insT</td>
<td>Frame-shift</td>
<td>Arg366fsX</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1056delG</td>
<td>2</td>
<td>Frame-shift</td>
<td>Gly352fsX</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1014insT</td>
<td>2</td>
<td>Missense</td>
<td>Lys338Lys</td>
<td>N</td>
</tr>
<tr>
<td>2 (NHL)</td>
<td>F</td>
<td>18</td>
<td>1099G&gt;C</td>
<td>1101G&gt;A</td>
<td>Missense</td>
<td>Missense</td>
<td>Arg366Thr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1101G&gt;A</td>
<td>2</td>
<td>Missense</td>
<td>Gly346Asp</td>
<td>N</td>
</tr>
</tbody>
</table>

**Figure 3.** TAL1 gene mutations in T-NHL patients. (A-B) Representative DNA
sequencing chromatograms of T-NHL genomic DNA samples showing
mutations in exon 2 of TAL1. WT: wild type. T-NHL: T-cell non-Hodgkin’s
lymphoma; TAL1 gene: T-cell acute leukemia 1 gene.

Table 4. T-ALL and T-NHL Patients’ characteristics and TAL1 exon 2 mutations.

<table>
<thead>
<tr>
<th>Patients’ characteristics</th>
<th>TAL1 wild type</th>
<th>TAL1 mutated</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-ALL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PLT, ×10^9/L (range)</td>
<td>166.41 ± 31.82</td>
<td>326.5 ± 176.5</td>
<td>0.177</td>
</tr>
<tr>
<td>WBC count, 10^9/L (range)</td>
<td>11.59 ± 4.36</td>
<td>8.0 ± 0.001</td>
<td>0.810</td>
</tr>
<tr>
<td>Age (years)</td>
<td>20.77 ± 2.17</td>
<td>25.0 ± 10.0</td>
<td>0.588</td>
</tr>
<tr>
<td>T-NHL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PLT, ×10^9/L (range)</td>
<td>178.83 ± 29.91</td>
<td>304 ± 0.01</td>
<td>0.165</td>
</tr>
<tr>
<td>WBC count, 10^9/L (range)</td>
<td>4.58 ± 0.98</td>
<td>6.5 ± 3.50</td>
<td>0.493</td>
</tr>
<tr>
<td>Age (years)</td>
<td>24.50 ± 6.5</td>
<td>24.50 ± 6.5</td>
<td>0.576</td>
</tr>
</tbody>
</table>

PLT: platelets; WBC: white blood cell; T-ALL: T-cell acute lymphoblastic
leukaemia; T-NHL: T-cell non-Hodgkin’s lymphoma; TAL1 gene: T-cell acute
leukemia 1 gene; P value: *rank sum test; #Fisher’s test.
are rarely identified in pediatric leukemias but represent one third of adult T-ALL cases [29]. Almost 25% of patients with T-ALL have tumor-specific rearrangements of the TALI gene [27-28]. Although TALI expression has not been observed in normal lymphocytes, TALI gene products are readily identified in leukemic cells that harbor a rearranged TALI allele. Hence, it has been suggested that ectopic expression of TALI promotes the T-ALL development. In our study, TALI exon 2 mutations were identified in 10% (n=20) of T-ALL patients analyzed. In contrast to previous findings [5], we have observed no male excess of TALI mutations in T-ALL patients. We detected excess of adolescent (12.5%, n=8) over adult (7.7%, n=13) cases of TALI exon 2 mutations in T-ALL patients. In contrast with our study, the TALI mutations were found more frequent in adult T-ALL [5]. Overall, TALI mutations were reported in this study, implicating exons 2. Among these, frame-shift and missense mutations accounted for, respectively, 44.4% and 55.6% of all TALI exon 2 mutations (Table 2, Figure 2). These mutations have not been reported previously.

In fact, T-NHL is usually differentiated from T-ALL by minimal or absent bone marrow and peripheral blood involvement, normal WBC count, normal hemoglobin levels and lack of organomegaly. Because this distinction does not apply in every case, a presence of less than 25% of blasts in the bone marrow is currently used to define T-NHL from T-ALL [4]. The relationship between TALI variants and T-NHL cancer risk has been investigated in several studies [4, 15, 24]. In our study, TALI exons 2 mutations were detected in 20% T-NHL patients analyzed. Again in contrast to a previous study [5], we found no difference in the rates of TALI mutated cases between male and female T-NHL patients from Libya. Missense and frame-shift mutations accounted for, respectively, 60% and 40% all TALI exon 2 mutations. In the present study, sequence of TALI exon 3 showed no sequence variation for the cancer patients analyzed. The absence of TALI exon 3 mutations in this study would have occurred due to the number of patient samples used.

Comparing the clinical features of cases showing TALI mutation positive and TALI mutation negative revealed that WBC counts the only significant variable between the two groups [5, 30]. Previously, it has been shown that the expression of T-cell markers CD3, CD4, and CD7 were less in cases showing TALI mutations compared to TALI mutations negative cases but they have not reached statistical significance. Moreover, it has been shown that the patients with TALI rearrangements at presentation are usually associated with high WBC count [5], CNS disease, immunophenotype of CD2+ and CD5+ [30]. In our study, we also found that TALI positive mutations were associated with higher PLT counts and WBC counts in cancer patients analyzed. Our study suggests that TALI mutations may cooperate with these genetic abnormalities during T-cell leukemogenesis.
Collectively, this study did not show any evidence of a significant correlation between TAL1 variants and increased risk of T-ALL and T-NHL. In spite of study is the first study to explore T-ALL and T-NHL in Libya at molecular level, the large sample size offers more reliable conclusions regarding T-ALL and T-NHL among Libyans. In our study, we found no difference in the rates of TAL1 mutated cases between male and female T-NHL patients from Libya. Together with previous investigations, our findings call for closer examination of entire coding region of TAL1 gene in T-ALL and T-NHL patients of both sexes in ethnically defined populations. Also, the present study makes it necessary to study other molecular abnormalities in T-ALL and T-NHL such as JAK1 mutation, p16 INK4a deletion, PHF6 mutation, Notch1 mutation, and TLX1 (HOX11) gene expression to explore whether they contribute to the high prevalence of T-ALL and T-NHL in Libyan patients with T-ALL and T-NHL.

Authors’ Contributions

Amal E. Elarifi, Othman A. El-Ansari and Mohamed A. Al-Griw substantially contributed to the conception and design of the study, acquisition, analysis and interpretation of data; all authors drafted the article and made critical revisions related to the intellectual content of the manuscript, and approved the final version of the article to be published.

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References


Prevalence of Mutations in TAL1 Gene in Individuals with T-ALL and T-NHL


Post-transplant Diabetes Mellitus (PTDM), a Retrospective Review Studying the Effect of Early Steroid Withdrawal in Libyan Recipients

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Abstract

Post-transplant diabetes mellitus (PTDM) is the most, and, frequent complication observed following solid organ transplantation. PTDM or the Kidney transplant recipients develop are also at increased risk of cardiovascular events and other adverse outcomes including infection, reduced patient survival, graft rejection, and accelerated graft loss compared with non-diabetics.

In general new onset of diabetes mellitus after transplantation has been reported to occur in 4% to 25% of renal transplant recipients, 2.5% to 25% of liver transplant recipients, and approximately 2% to 53% of all people. 230 renal transplant recipients with functioning grafts were used in this study. This study took about 5 years in order to screen the incidences of PTDM and other risk factors.

The aim is to study the incidence of new-onset of diabetes mellitus in kidney transplant recipients and correlate it with protocol of withdrawing steroids after one month after transplantation.

The results confirm the importance of corticosteroids in the development of post transplantation new onset of diabetes mellitus in Libyan population. The data indicates that Libyan male patients are more vulnerable to PTDM than Libyan female patients.

Keywords: PTDM, transplant recipients; diabetes mellitus; corticosteroids; corticosteroids receptor immunosuppressive.

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Introduction

Corticosteroids are a class of steroids that are produced in the adrenal cortex of humans. They are involved in a wide range of physiological processes including immune response, stress response and regulation of inflammation, carbohydrate metabolism, protein catabolism, behavior and blood electrolyte levels (Nussey et al., 2001; Blackburn et al., 2002).

Corticosteroids are form an essential compound of most immunosuppressor regions. They are nowadays used in renal transplantation because of their efficacy in reducing acute rejection and improving graft survival. Steroids, however, are associated with numerous mid effects that lead to increased patients morbidities mortalities (Veenstra et al., 1999).

High incidences of PTDM are associated with the type of initial maintenance immunosuppression, race, ethnicity, obesity and hepatitis C infection. It is a strong independent predictor of graft failure and mortality. Efforts should be made to minimize the risk of its important implication (Kasiske et al., 2003; Cole et al., 2008).

Efforts to reduce PTDM, while maintaining low rates of AR, will have the potential of improving long-term outcomes. Therefore, the new studies have shown that early steroid withdrawal have limited impact on PTDM, but it is associated with a higher incidence of AR (Vincent et al., 2008; Woodle et al., 2008). It is known that PTDM is associated with steroids as well as calcineurin inhibitors, especially tacrolimus (Chadban et al., 2008).
The number of acute rejection episodes in early post-transplant has declined dramatically and graft survival, particularly during the first year post-transplant, has improved substantially (Schweitzer et al., 1991). It is clear that in large part, these improvements are consequences of the use of better immunosuppressive protocols and employing newer, more potent immunosuppressive drugs. High patient mortality, however, continues to be the major threat to the success of renal transplantation (Vanrenterghem et al., 2005). Because the excess of mortality in transplant recipients is largely due to cardiovascular causes (Benfield et al., 2010), searching for variables associated with increased cardiovascular risk and correcting those variables are critically important. Previous studies have identified several cardiovascular risk factors in patients with end-stage renal disease and in transplant patients (Hocker et al., 2010; Pelletier et al., 2006). However, it has also been pointed out that the cardiovascular mortality of patients with kidney disease is much higher than that of patients without kidney problems who have a similar risk profile (Sola et al., 2002). The latter results suggest that other cardiovascular risk factors need to be considered in patients with kidney disease. One of those factors is likely to be insulin resistance that commonly occurs in patients receiving immunosuppressive medications and is clearly associated with increased cardiovascular risk (Gregoor et al., 2002; Boletis et al., 2001; Ahsan et al., 1999). A plasma or serum glucose level lower than 140mg/dL is normal and requires no follow-up. If the glucose level is 140mg/dl or higher after a three hour OGTT (Oral Glucose Tolerance Test) is performed, it is considered normal blood glucose level reading and without fasting first, a reading of under 200 mg/dl is considered normal. A level of over 200 mg/dl, especially with symptoms of frequent urination, excessive thirst, etc. will indicate a strong possibility of diabetes.

**Material and Methods**

Blood samples (5ml) of 230 renal transplant recipients with functioning grafts, were collected over period of five years in Kidney Transplant Center in Tripoli, Libya.

The following tests were performed; urine examination for glucose, fasting glucose (FBG), Oral Glucose Tolerance Test (OGTT) and glucosalated heamoglobin A one C (HbA1C). Assay was done at least weekly for four weeks and every three months for one year.

**Study Population**

Adult recipients (≥18 years of age) of a first deceased or living kidney donor only transplant were considered eligible for the study. Only non-diabetic patients
were eligible; all patients were required to have a normal 2-hour 75-g OGTT (<7.8 mmol/l) performed within 1 month of the date of transplantation. Patients with an OGTT of 7.8 to 11.0 mmol/l were classified as having impaired glucose tolerance and were included in the study, while patients with an OGTT >11.0 mmol/l were excluded. In addition, patients were excluded if they had pre-transplant Panel Reactive Antibody >20%, or if they received a zero A, B or DR mismatched kidney. Patients were also excluded if they were unable to provide informed consent or were hepatitis C antibody positive.

**Diagnosis of Diabetes and Glucose Intolerance**
1- Diabetes symptoms with randomized plasma blood glucose ≥200 mg/dL (11.1 mmol/L) or fasting plasma glucose (FPG) (at least 8 hours fast) ≥126 mg/dL (7.0 mmol/L).
2. Fasting intolerance FPG ≥110 mg/dl (6.1 mmol/l) and < 126 mg/dl (7.0 mmol/l).
3. Oral test for glucose intolerance (glucose load at 75g of glucose dissolved in water) 2-hour plasma glucose ≥140 mg/dL (7.8 mmol/L) and < 200 mg/dL (11.1 mmol/L).

**Results**

The results of 230 renal transplant recipients with functioning grafts, performed between 2004 to 2009, were screened for the incidences of PTDM and other risk factors, including gender, age, tissue typing type of immunosuppression treatment, presence of hepatitis and Clinical impact of PTDM (Figure 1).

This first study on Libyan population shows that the males patients are more affected with Post Transplantation DM than females (Figure 2) especially the age group between 46-55 age old men, the Libyan women patients are more affected than women with new onset diabetes mellitus after treated with steroid hormone (Corticosteroids) and Figure 2 show that the risk factor of onset diabetes mellitus after post transplantation is very few in Libyan women with group age 26-35.

PTDM adversely affects long-term allograft survival. In one study, for example, graft survival at 12 years was 48 and 70 percent in those with and without PTDM, respectively.

The incidences of PTDM after 1, 3, and 5 years post transplantation in these patients were < 4%. Taking into account the risk factors with regard to gender or presence of hepatitis, B and/or C, 252 post kidney transplantation patients were checked for diabetes by measuring their fasting plasma glucose.
Post-transplant Diabetes Mellitus (PTDM), a Retrospective Review

Accordingly, 8 patients (3.2%) were found to have diabetes millets (plasma glucose >126 mg%) and 14 patients (5.5%) having impaired fasting plasma glucose (> 110 mg% and < 126 mg%)

Figure 1. Risk factors for post-transplant diabetes development (Patients with impaired fasting glycaemia).

Discussion and Conclusion

Early corticosteroids withdrawal play a role in reducing the development of post transplantation new onset diabetes mellitus in Libyan population. It is clear, however, that the field is at an exciting stage. The next few years should provide a big step forward in our understanding of how these important anti-inflammatory molecules exert their effects, with concomitant advances in the clinical treatment of inflammatory disease.

In the present study, we evaluated the incidence of PTDM in a Libyan group of renal allograft recipients with transplants in a single institution (Libyan National Organ Transplantation Program), and treated with uniform corticosteroids immunosuppressive protocols. The incidences of PTDM reported here is similar to that incidences reported in other studies. However, there is a significant variability in the reported incidences of PTDM, most likely because of at least three reasons. First, the criteria used to diagnose PTDM are quite variable among studies. Second, variability in the immunosuppressive protocols used in different transplant centers will have an impact in the incidence of PTDM. For example,
the incidence of PTDM is significantly higher in transplant recipients treated with tacrolimus than in those treated with Corticosteroids (Shiraswamy et al., 2016; Wallia et al., 2016).

Figure 2. Patients with Post Transplantation DM transplant recipients older than 45 years of age were 2.2 times more likely to develop PTDM than those younger than 45 at the time of transplantation (P< 0.0001).

The development of post-transplant diabetes mellitus PTDM is associated with a high risk of complications, such as infections and cardiovascular disease. Identifying patients at high risk of developing PTDM by close monitoring of glucose level and prompt therapy of hyperglycemia are warranted. One of these modifiable risk factors is Corticosteroid therapy. Comparing our study with others surely showed that early corticosteroids withdrawal has a role in reducing the development of post transplantation new onset diabetes mellitus (Penformis and Kury-Paulin, 2006).

It was suggested in the literature that African and Hispanics are at increased risk for developing PTDM compared to whites. Thus, the risk of developing PTDM as defined by the 2003 International Guidelines was double in Africans compared to whites. Eighteen similar data from the USRDS demonstrated that PTDM was more common among African Americans (RR=1.68, P< 0.0001) and Hispanics (RR=1.35, P< 0.0001) compared with Caucasians.
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This study shows that the most important modifiable risk factors of development post-transplant diabetes mellitus PTDM is corticosteroid therapy. We show here that early corticosteroids withdrawal has a crucial role in reducing the development of post transplantation new onset diabetes mellitus and that the Libyan women interact more positively with corticosteroids than Libyan men in avoiding development post-transplant diabetes mellitus PTDM.

Authors’ Contributions

The corresponding author Dr. Abdulhafid Shebani certifies that all co-authors have approved and agreed to the contents of manuscript and that the submitted work has not been considered for publication or published previously.

References


Post-transplant Diabetes Mellitus (PTDM), a Retrospective Review

On Solving Some Fifth Order Nonlinear PDEs Using The Modified Kudryashov Method

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Abstract

In this paper, the modified Kudryashov method was applied to construct the exact travelling wave solutions for some fifth order nonlinear partial differential equations (PDEs), namely, the Kaup-Kupershmidt, the Ito, the Caudrey-Dodd-Gibbon, the Lax and the Sawada-Kotera equations.

Keywords: Modified Kudryashov method; Nonlinear PDEs; Exact solutions.

المستخلص

في هذه الورقة طبقت طريقة قدير شوف المطورة لبناء حلول موجية متحركة تامة لبعض المعادلات التفاضلية الجزئية غير الخطية من الرتبة الخامسة وهي معادلات كوب-كويريشميت ، وأيتو، وكودري-تود - جيبون، وللخوو، وسواتا كوتيرا.

Introduction

In the last four decades or so, seeking exact solutions of nonlinear PDEs has been of great importance, since the nonlinear complex physical phenomena related to the nonlinear PDEs arise in many fields of physics, mechanics, biology, chemistry and engineering. The investigation of exact solutions of nonlinear PDEs, as mathematical models of the phenomena, will help us to understand the mechanism that governs.
these physical models or provide better understanding of the problems and the possible applications. To these ends, a vast variety of powerful and direct methods for finding the exact significant solutions of nonlinear PDEs have been derived, such as the inverse scattering transform [1], the Hirota method [2], the truncated Painleve expansion method [4], the Backlund transform method [1], the simplest equation method [6], the Jacobi elliptic function method [7], the tanh-function method [12], the modified simple equation method [3], the Kudryashov method [5,13,14], the multiple exp-function algorithm method [8], the transformed rational function method [9], the Frobenius decomposition technique [10], the local fractional variation iteration method [17] and the local fractional series expansion method [18] and so on.

The objective of this paper is to demonstrate the efficiency of the modified Kudryashov method for finding exact solutions of some nonlinear evolution equations in the mathematical physics, namely, the Kaup-Kupershmidt, the Ito, the Caudrey-Dodd-Gibbon, the Lax and the Sawada-Kotera equations.

**Description of the Modified Kudryashov Method**

Suppose we have a nonlinear evolution equation in the form

\[ F(u, u_t, u_x, u_{xx}, \ldots) = 0 \]  

Where \( F \) is polynomial in \( u(x,t) \) and its partial derivatives in which the highest order derivatives and nonlinear terms are involved. In the following, we give the main steps of this method [11]:

**Step 1.** Using the wave transformation

\[ u(x,t) = u(\xi), \quad \xi = kx + \omega t \]  

To reduce Eq. (1) to the following ODE:

\[ P(u, u', u'', \ldots) = 0, \]  

Where \( P \) is a polynomial in \( u(\xi) \) and its total derivatives, while \( k, \omega \) are constants and the prime notation in (3) denotes differentiation with respect to \( \xi \).
On Solving Some Fifth Order Nonlinear PDEs Using the Modified Kudryashov Method

**Step 2.** We suppose that Eq. (3) has the formal solution

\[ u(\xi) = \sum_{n=0}^{N} a_n Q^n(\xi), \]

(4)

where \( a_n \ (n = 0, 1, ..., N) \) are constants to be determined, such that \( a_N \neq 0 \), and \( Q(\xi) \) is the solution of the equation

\[ Q'(\xi) = \left[ Q^2(\xi) - Q(\xi) \right] \ln(a) \]

(5)

Eq. (5) has the solutions

\[ Q(\xi) = \frac{1}{1 \pm a \xi} \]

(6)

Where \( a > 0, \ a \neq 1 \) is a number. If \( a = e \), then we have the modified Kudryashov method which has been applied by many authors, see for example [5].

**Step 3.** We determine the positive integer \( N \) in Eq. (4) by considering the homogeneous balance between the highest order derivatives and the nonlinear terms in Eq. (3).

**Step 4.** Substitute Eq. (4) along with Eq. (5) into Eq. (3), we calculate all the necessary derivatives \( u', u'', ... \) of the function \( u(\xi) \). As a result of this substitution, we get a polynomial of \( Q'(\xi) \), \( i = 0, 1, 2, ... \). In this polynomial we gather all terms of same powers of \( Q'(\xi) \) and equating them to zero, we obtain a system of algebraic equations which can be solved by the Maple or Mathematica to get the unknown parameters \( a_n \ (n = 0, 1, ..., N) \), \( k \) and \( \omega \). Consequently, we obtain the exact solutions of Eq. (1).

**Remark 1.** The obtained solutions can depended on the symmetrical hyperbolic Lucas functions and Fibonacci functions proposed by Stakhov and Rozin [15]. The symmetrical Lucas sine, cosine, tangent and cotangent functions are respectively, defined as
Also, these functions satisfy the following formulas:

\[
[cLs(\xi)]^2 - [sLs(\xi)]^2 = 4
\]  

(8)

\[
[cFs(\xi)]^2 - [sFs(\xi)]^2 = \frac{4}{5}
\]  

(9)

The obtained solutions in this paper can be obtained in terms of the symmetrical hyperbolic Lucas functions.

**Applications**

In this section, we apply the modified Kudryashov method to find the exact solutions of the following nonlinear PDEs:

**Example 1. The Kaup-Kupershmidt (KK) Equation**

This equation is well known [16] and has the form

\[
\frac{\partial^3 u}{\partial \xi^3} + 20uu^2u_x + 25uu_{xx} + 10uu_{\xi} + uu_{\xi\xi} = 0.
\]

(10)

Let us now solve equation (10) using the modified Kudryashov method. To this end, we use the wave transformation (2) to reduce equation (10) to the following ODE:

\[
ou' + 20ku^2u' + 25k^3u'^3 + 10k^3uu^{(3)} + k^5u^{(5)} = 0.
\]

(11)

Balancing \(u^{(5)}\) with \(u'\) yields \(N = 2\). Consequently, equation (11) has the formal solution

\[
u = a_0 + a_1Q + a_2Q^2
\]

(12)
On Solving Some Fifth Order Nonlinear PDEs Using the Modified Kudryashov Method

where $a_0, a_1$, and $a_2$ are constants to be determined such that $a_2 \neq 0$. From equation (12), we get

$$u' = (\ln a)(a_1 + 2QA_2)Q(Q - 1),$$

(13)

$$u'' = (\ln a)^2Q(Q - 1)[(-1 + 2Q)a_1 + 2Q(3Q - 2)a_2],$$

(14)

$$u^{(3)} = (\ln a)^3Q(Q - 1)[(1 - 6Q + 6Q^2)a_1 + 2Q(4 - 15 + 12Q^2)a_2],$$

(15)

$$u^{(4)} = (\ln a)^4Q(Q - 1)[(-1 + 14Q - 36Q^2 + 24Q^3)a_1 + 2Q(-8 + 57Q - 108Q^2 + 60Q^3)a_2],$$

(16)

$$u^{(5)} = (\ln a)^5Q(Q - 1)[(1 - 30Q + 150Q^2 - 240Q^3 + 120Q^4)a_1 + 2Q(16 - 195Q + 660Q^2 - 840Q^3 + 360Q^4)a_2]$$

(17)

Substituting (12)-(17) into (11) and equating all the coefficients of powers of $Q(\xi)$ to zero, we obtain algebraic system of equations, on solving the obtained algebraic equations using the Maple or Mathematica, we get the following results:

**Case 1.**

$$a_0 = -k^2(\ln a)^2, a_1 = 12k^2(\ln a)^2, a_2 = -12k^2(\ln a)^2, \omega = -11k^5(\ln a)^4.$$  

(18)

From (6), (7), (12), (18), we obtain the following exact solutions of Eq. (10)

$$u_1(x,t) = -k^2(\ln a)^2 + 12$$

$$\left(\frac{k \ln(a)}{cLs\left(\frac{\xi}{2}\right)}\right)^2,$$

(19)

$$u_2(x,t) = -k^2(\ln a)^2 - 12$$

$$\left(\frac{k \ln(a)}{sLs\left(\frac{\xi}{2}\right)}\right)^2,$$

(20)
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Case 2.

\[ a_0 = \frac{1}{8} k^2 (\ln a)^2, \quad a_1 = \frac{3}{2} k^2 (\ln a)^2, \quad a_2 = -\frac{3}{2} k^2 (\ln a)^2, \quad \omega = -\frac{1}{16} k^5 (\ln a)^4. \]  \hspace{1cm} (21)

\[ u_3(x,t) = -\frac{1}{8} k^2 (\ln a)^2 + \frac{3}{2} \left( \frac{k \ln(a)}{cLs \left( \frac{\xi}{2} \right)} \right)^2, \]  \hspace{1cm} (22)

\[ u_4(x,t) = -\frac{1}{8} k^2 (\ln a)^2 - \frac{3}{2} \left( \frac{k \ln(a)}{sLs \left( \frac{\xi}{2} \right)} \right)^2. \]  \hspace{1cm} (23)

**Example 2. The Ito Equation**

This equation is well known [16] and has the form

\[ u_t + 2u^2u_x + 6uu_{xx} + 3uu_{xxx} + uu_{xxxx} = 0. \]  \hspace{1cm} (24)

Let us solve equation (24) by using the modified Kudryashov method. To this end, we use the wave transformation (2) to reduce equation (24) to the following ODE:

\[ \omega u' + 2ku^2u' + 6k^3 uu' + 3k^3 uu^{(3)} + k^5 u^{(5)} = 0. \]  \hspace{1cm} (25)

Balancing \( u^{(5)} \) with \( u^2u' \) yields \( N = 2 \). Consequently, equation (24) has the formal solution (12). Substituting (12)-(17) into (24) and equating all the coefficients of powers of \( Q(\xi) \) to zero, we obtain algebraic system of equations, on solving the obtained algebraic equations using the Maple or Mathematica, we get the following result:

\[ a_0 = -\frac{5}{2} k^2 (\ln a)^2, \quad a_1 = 30k^2 (\ln a)^2, \quad a_2 = -30k^2 (\ln a)^2, \quad \omega = -6k^5 (\ln a)^4 \]  \hspace{1cm} (26)
On Solving Some Fifth Order Nonlinear PDEs Using the Modified Kudryashov Method

From (6), (7), (12), (26), we obtain the following exact solutions of Eq. (25)

\[ u_1(x,t) = \frac{-5}{2} k^2 (\ln a)^2 + 30 \left( \frac{k \ln a}{cLs} \right)^2, \]

\[ u_2(x,t) = \frac{-5}{2} k^2 (\ln a)^2 - 30 \left( \frac{k \ln a}{sLs} \right)^2, \]

Example 3. The Caudrey-Dodd-Gibbon Equation (CDG)
This equation is well known [16] and has the form:

\[ u_t + 180 u^2 u_x + 30 uu_x + 30 uu_{3x} + u_{5x} = 0. \]

Let us solve equation (29) by the modified Kudryashov method. To this end, we use the wave transformation (2) to reduce equation (29) to the following ODE:

\[ \omega u' + 180k u^2 u' + 30k^3 u'' + 30k^3 u^{(3)} + k^5 u^{(5)} = 0. \]

Balancing \( u^{(5)} \) with \( u^2 u' \) yields \( N = 2 \). Consequently, equation (29) has the formal solution (12). Substituting (12)-(17) into (29) and equating all the coefficients of powers of \( Q(\xi) \) to zero, we obtain algebraic system of equations, on solving the obtained algebraic equations using the Maple or Mathematica, we get the following results:

Case 1.

\[ a_1 = k^2 (\ln a)^2, \quad a_2 = -a_1, \quad \omega = -k^5 (\ln a)^4 - 180ka_0^2 - 30k^3 a_0 (\ln a)^2. \]

In this case, we deduce the following exact solutions of Eq. (29)
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\[ u_1(x,t) = a_0 + \left( \frac{k \ln a}{cLs\left( \frac{x}{2} \right)} \right)^2, \quad (32) \]

\[ u_2(x,t) = a_0 - \left( \frac{k \ln a}{sLs\left( \frac{x}{2} \right)} \right)^2, \quad (33) \]

**Case 2.**

\[ a_0 = -\frac{1}{6} k^2 (\ln a)^2, \quad a_1 = 2k^2 (\ln a)^2, \quad a_2 = -2k^2 (\ln a)^2, \quad \omega = -k^5 (\ln a)^4. \quad (34) \]

In this case, we deduce the following exact solutions of Eq. (29)

\[ u_3(x,t) = \frac{-1}{6} k^2 (\ln a)^2 + 2 \left( \frac{k \ln a}{cLs\left( \frac{x}{2} \right)} \right)^2, \quad (35) \]

\[ u_4(x,t) = \frac{-1}{6} k^2 (\ln a)^2 - 2 \left( \frac{k \ln a}{sLs\left( \frac{x}{2} \right)} \right)^2, \quad (36) \]

**Example 4. The Lax Equation**

This equation is well known [16] and has the form:

\[ u_t + 30u^2u_x + 20uu_{xx} + 10uu_{xxx} + u_{xxx} = 0. \quad (37) \]

Let us solve equation (37) by the modified Kudryashov method. To this end, we use the wave transformation (2) to reduce equation (37) to the following ODE:
On Solving Some Fifth Order Nonlinear PDEs Using the Modified Kudryashov Method

\[ au' + 30ku^2u' + 20k^3u'u'' + 10k^3uu'' + k^5u^{(5)} = 0. \]  (38)

Balancing \( u^{(5)} \) with \( u'u' \) yields \( N = 2 \). Consequently, equation (37) has the formal solution (12). Substituting (12)-(17) into (37) and equating all the coefficients of powers of \( Q(\xi) \) to zero, we obtain algebraic system of equations. On solving the obtained algebraic equations using the Maple or Mathematica, we get the following results:

**Case 1.**

\[ a_1 = 2k^2(\ln a)^2, \quad a_2 = -a_1, \quad \omega = -k^5(\ln a)^4 - 30ka_0^2 - 10k^3a_0(\ln a)^2. \]  (39)

In this case, we deduce the following exact solutions of Eq. (37)

\[ u_1(x,t) = a_0 + 2 \left( \frac{k \ln a}{cLs \left( \frac{\xi}{2} \right)} \right)^2, \]  (40)

\[ u_2(x,t) = a_0 - 2 \left( \frac{k \ln a}{sLs \left( \frac{\xi}{2} \right)} \right)^2. \]  (41)

**Case 2.**

\[ a_0 = \frac{-1}{2} k^2(\ln a)^3, \quad a_1 = 6k^2(\ln a)^2, \quad a_2 = -6k^2(\ln a)^2, \quad \omega = \frac{-7}{2} k^5(\ln a)^4. \]  (42)

In this case, we deduce the following exact solutions of Eq. (37)

\[ u_3(x,t) = \frac{-1}{2} k^2(\ln a)^2 + 6 \left( \frac{k \ln a}{cLs \left( \frac{\xi}{2} \right)} \right)^2, \]  (43)
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\[ u_a(x,t) = \frac{-1}{2} k^2 (\ln a)^2 - 6 \left( \frac{k \ln a}{sLs \left( \frac{\xi}{2} \right)} \right)^2, \quad (44) \]

**Example 5. The Sawada-Kotera (SK) Equation**

This equation is well known [16] and has the form:

\[ u_t + 5u^2u_x + 5uu_{xx} + 5uu_{x} + u_{x} = 0. \quad (45) \]

Let us solve equation (45) by the modified Kudryashov method. To this end, we use the wave transformation (2) to reduce equation (45) to the following ODE:

\[ \omega u' + 5ku^2u' + 5k^3uu'' + 5k^3uu^{(3)} + k^5u^{(5)} = 0. \quad (46) \]

Balancing \( u^{(5)} \) with \( u^2u' \) yields \( N = 2 \). Consequently, equation (46) has the formal solution (12). Substituting (12)-(17) into (46) and equating all the coefficients of powers of \( Q(\xi) \) to zero, we obtain algebraic system of equations. On solving the obtained algebraic equations using the Maple or Mathematica, we get the following results:

**Case 1.**

\[ a_1 = 6k^2 (\ln a)^2, \quad a_2 = -a_1, \quad \omega = -k^3 (\ln a)^4 - 5ka_0^2 - 5k^3a_0 (\ln a)^2. \quad (47) \]

In this case, we deduce the following exact solutions of Eq. (45)

\[ u_1(x,t) = a_0 + 6 \left( \frac{k \ln a}{cLs \left( \frac{\xi}{2} \right)} \right)^2, \quad (48) \]
On Solving Some Fifth Order Nonlinear PDEs Using the Modified Kudryashov Method

\[ u_2(x,t) = a_0 - 6 \left( \frac{k \ln a}{sLs \left( \frac{\xi}{2} \right)} \right)^2, \quad (49) \]

**Case 2.**

\[ a_0 = -k^2 (\ln a)^2, \quad a_1 = 12k^2 (\ln a)^2, \quad a_2 = -a_1, \quad \omega = -k^3 (\ln a)^4. \quad (50) \]

In this case, we deduce the following exact solutions of Eq. (45)

\[ u_3(x,t) = -k^2 (\ln a)^2 + 12 \left( \frac{k \ln a}{cLs \left( \frac{\xi}{2} \right)} \right)^2, \quad (51) \]

\[ u_4(x,t) = -k^2 (\ln a)^2 - 12 \left( \frac{k \ln a}{sLs \left( \frac{\xi}{2} \right)} \right)^2. \quad (52) \]

**Physical Explanations of the Obtained Solutions**

In this section we have presented some graphs of these solutions by taking suitable values of involved unknown parameters to visualize the underlying mechanism of the original equations. The solution obtained in this paper are bell-shaped soliton solutions and singular bell-shaped soliton solution. Using mathematical software Maple or Mathematica, the plots of some obtained solutions of equations (19) and (28) have been shown in Figs.1-2.
Fig. 1. The plot of solution (19) when $k = 1, \omega = 1, \alpha = 2$.

Fig. 2. The plot of solution (28) when $k = 2, \omega = 1, \alpha = 3$. 

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Conclusion

In summary, we have presented the modified Kudryashov method and used it to construct more general exact solutions of nonlinear PDE's with the aid of Maple 14. This method provides a powerful mathematical tool for obtaining more general exact solutions of many nonlinear PDE's in mathematical physics. Applying this method to the indicated equations, we have successfully obtained many new exact travelling wave solutions. To our knowledge, these solutions have not been reported in the former literature. Furthermore, this method is valid for a large number of nonlinear equations with variable coefficients. Finally, all solutions obtained in this article have been checked with the Maple 14 by putting them back into the original equation.

References


On Extremal Topology

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Abstract

Extremal topology was defined on an arbitrary set $X$ as a maximal non-discrete topology [2]. In this paper we will prove that every extremal topology $\tau$ on a set $X$ has to be of the form $\tau = P(X \setminus \{x\}) \cup \{\{x\} \cup F : F \in \mathcal{F}\}$, for some $x \in X$ and some ultrafilter $\mathcal{F}$ in $X \setminus \{x\}$. Where $P(X \setminus \{x\})$ is the power set of $X \setminus \{x\}$. We also show that if $\mathcal{F}$ is a free ultrafilter then $(X, \tau)$ is a $T_4$ space.

Keywords: Extremal topology; Door Space; $T_4$ Space.

Preliminaries

If $X$ is a non-empty set, a non-empty collection $\mathcal{F}$ of subsets of $X$ is called a filter in $X$ if (i) $\emptyset \notin \mathcal{F}$, (ii) if $F_1, F_2 \in \mathcal{F}$ then $F_1 \cap F_2 \in \mathcal{F}$, (iii) if $F \in \mathcal{F}$ and $G \subseteq X$ with $F \subseteq G$ then $G \in \mathcal{F}$.

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A filter \( \mathcal{F} \) on \( X \) is said to be free filter provided \( \bigcap_{E \in \mathcal{F}} E = \emptyset \) otherwise it is called a fixed filter. A filter \( \mathcal{F} \) is called an ultrafilter if it is a maximal filter; that is if \( \mathcal{F} \) is a filter containing \( \mathcal{F} \), then \( \mathcal{F} = \mathcal{G} \). A filter \( \mathcal{F} \) is an ultrafilter in \( X \) if and only if for any \( E \subseteq X \) either \( E \in \mathcal{F} \) or \( X \setminus E \in \mathcal{F} \) and an ultrafilter \( \mathcal{F} \) is fixed ultrafilter if and only if there exists, \( y \in X \) such that \( \bigcap_{E \in \mathcal{F}} E = \{y\} \). A collection \( \mathcal{D} \) of subsets of \( X \) is a filter base for a filter in \( X \) if and only if: (i) \( \emptyset \notin \mathcal{D} \), (ii) For any \( D_1, D_2 \in \mathcal{D} \) there exists \( D_3 \in \mathcal{D} \) with \( D_3 \subseteq D_1 \cap D_2 \). Every filter is contained in an ultrafilter see [1] and [4]. A topological space \( X \) is said to be a door space if any subset of \( X \) is either open or closed [1]. A topological space \( X \) is called a \( T_1 \) space if every one-point set is either open or closed. Clearly if \( X \) is a door space then \( X \) is a \( T_1 \) space.

In [2] extremal topology was defined, and it was proved that for any \( x, y \in X, x \neq y, \tau_{(x,y)} = \mathcal{P}(X \setminus \{x\}) \cup \{\{x\} \cup A: A \subset \mathcal{P}(X \setminus \{x\}), y \in A\} \) is an extremal topology and if \( X \) is finite then every extremal topology on \( X \) has to be of the form \( \tau_{(x,y)} \) for some \( x, y \in X, x \neq y \). In this paper we will generalize Theorem 1-2 and Theorem 2-1 of [2] and derive some other properties of extremal spaces.

The Main Results

We first give the following Theorem

**Theorem 1:**

If \( X \) is a non-empty set, \( x \in X \) and \( \tau = \mathcal{P}(X \setminus \{x\}) \cup \{\{x\} \cup F : F \in \mathcal{F}\} \) for some filter \( \mathcal{F} \) in \( X \setminus \{x\} \) then:

a) \( \tau \) is a topology on \( X \).

b) \((X, \tau)\) is a normal space.

c) \((X, \tau)\) is a door space and hence a \( T_1 \) space.

d) If \( \mathcal{F} \) is a free filter in \( X \setminus \{x\} \), then \((X, \tau)\) is a Hausdorff space and hence a \( T_4 \) space.

**Proof:**

a) Is trivial.

b) Let \( A, B \) be any two disjoint closed subsets. Then we have two cases:

   case(i): If \( x \notin A \cup B \) then \( A, B \) are two disjoint open sets.

   case(ii): If \( x \in A \cup B \), say \( x \in A, x \notin B \). Then \( B \) is a clopen (closed and open) subset and so \( B^c \) and \( B \) are two disjoint open sets containing \( A \) and \( B \) respectively. Hence \( X \) is a normal space.
On External Topology

c) Let $A$ be any subset of $X$. If $x \notin A$, then $A$ is an open set. If $x \in A$, then for any $y \notin A$, $\{y\}$ is an open set containing $y$ and disjoint from $A$. Hence $A$ is a closed set. So $(X, \tau)$ is a door space.

d) If $\mathcal{F}$ is a free filter in $X\setminus\{x\}$, $y, z \in X, y \neq z$. Then if $y, z \in X\setminus\{x\}$, the sets $\{y\}, \{z\}$ are two disjoint open sets containing $y$ and $z$ respectively. If $z = x, y \neq x$, then since $\mathcal{F}$ is a free filter in $X\setminus\{x\}$, so there exists $F \in \mathcal{F}$ with $y \notin F$ and hence $\{x\} \cup F, \{y\}$ are two disjoint open sets containing $x$ and $y$ respectively. Therefore $X$ is a Hausdorff space and hence by (b) above $X$ is a $T_4$ space.

The following Theorem generalizes both 1-2 and 2-1 of [2]

**Theorem 2 [3]:**

A topology $\tau$ on $X$ is an extremal if and only if there exists $x \in X$ such that $\tau = P(X\setminus\{x\}) \cup \{x\} \cup \{F: F \in \mathcal{F}\}$ for some ultrafilter $\mathcal{F}$ in $X\setminus\{x\}$.

**Proof:**

$\Rightarrow$ Suppose $\tau$ is an extremal topology, then there exists $x \in X$ with $\{x\} \notin \tau$.

Let $\mathcal{D} = \{D \subseteq X\setminus\{x\}: \{x\} \cup D \in \tau\}$, then $\mathcal{D}$ is a filter base for a filter $\mathcal{G}$ in $X\setminus\{x\}$, and if $\bigcap_{D \in \mathcal{D}} D = \emptyset$ then $\mathcal{G}$ is a free filter while if $\bigcap_{D \in \mathcal{D}} D \neq \emptyset$ then $\mathcal{G}$ is a fixed filter. Let $\mathcal{F}$ be an ultrafilter in $X\setminus\{x\}$ containing $\mathcal{G}$ and $\tau^*$ be the topology generated by the collection $\tau \cup \{\{x\} \cup F: F \in \mathcal{F}\}$. Since $\{x\} \notin \tau^*$, then $\tau^*$ is not discrete and since $\tau \subseteq \tau^*$ and $\tau$ is an extremal so $\tau = \tau^*$ and hence $\{\{x\} \cup F: F \in \mathcal{F}\} \subseteq \tau$. Also since $\tau$ is extremal $A \in \tau$ for any $A \subseteq X\setminus\{x\}$, otherwise

$$\tau < A > = \{U \cup (V \cap A): U, V \in \tau\}$$

is not a discrete topology containing $\tau$ and with $A \in \tau < A >$. Which is a contradiction. Hence

$$P(X\setminus\{x\}) \cup \{\{x\} \cup F: F \in \mathcal{F}\} \subseteq \tau.$$ 

If $U \in \tau$, then if $x \notin U$ we then have $U \subseteq X\setminus\{x\}$ and so

$$U \in P(X\setminus\{x\}) \cup \{\{x\} \cup F: F \in \mathcal{F}\}.$$ 

If $x \in U$, then since $\mathcal{F}$ is an ultrafilter so either $U\setminus\{x\} \in \mathcal{F}$ or $X\setminus U \in \mathcal{F}$. If $U\setminus\{x\} \in \mathcal{F}$ then $U \in P(X\setminus\{x\}) \cup \{\{x\} \cup F: F \in \mathcal{F}\}$.

If $X\setminus U \in \mathcal{F}$ then $\{x\} \cup (X\setminus U) \in \tau$ and so $\{x\} = U \cap [\{x\} \cup (X\setminus U)] \in \tau$, which is a contradiction. So we have

$$\tau = P(X\setminus\{x\}) \cup \{\{x\} \cup F: F \in \mathcal{F}\}.$$
\(\Leftrightarrow\) Suppose \(\tau = P(X\{x\}) \cup \{x\} \cup F: F \in \mathcal{F}\) for some \(x \in X\) and some ultrafilter \(\mathcal{F}\) in \(X\{x\}\). To show that \(\tau\) is an extremal. Let \(\hat{\tau}\) be a non-discrete topology with \(\tau \subset \hat{\tau}\) and \(\tau \neq \hat{\tau}\). Then there exists \(w \in \hat{\tau}\) with \(w \notin \tau\).

If \(x \notin w\) then \(w \in P(X\{x\}) \subset \tau\) and we have a contradiction.

If \(x \in w\), then since \(\mathcal{F}\) is an ultrafilter in \(X\{x\}\), so either \(w\{x\} \in \mathcal{F}\) or \(X\{x\} \in \mathcal{F}\). If \(w\{x\} \in \mathcal{F}\), then \(w \in \tau\) and we have a contradiction. If \(X\{x\} \in \mathcal{F}\) then \(\{x\} \cup (X\{x\}) \in \tau \subset \hat{\tau}\), and so \(w \cap \{x\} \cup (X\{x\}) = \{x\} \in \hat{\tau}\), a contradiction since \(\hat{\tau}\) is not discrete so \(\tau = \hat{\tau}\) and hence \(\tau\) is an extremal topology on \(X\).

**Corollary 3:**
If \(\tau = P(X\{x\}) \cup \{x\} \cup F: F \in \mathcal{F}\) is an extremal topology and \(\mathcal{F}\) is a fixed ultrafilter in \(X\{x\}\), then \(\tau = \tau_{\{x,y\}}\) for some \(y \neq x\).

**Proof:**
If \(\mathcal{F}\) is a fixed ultrafilter, then there is \(y \in X\{x\}\) with \(\bigcap_{F \in \mathcal{F}} F = \{y\}\). So \(\tau = P(X\{x\}) \cup \{x\} \cup F: F \in \mathcal{F}\) = \(P(X\{x\}) \cup \{x\} \cup F: y \in F, F \in \mathcal{F}\) = \(\tau_{\{x,y\}}\).

The following two corollaries are consequences of Theorem 2.

**Corollary 4:**
If \(\tau\) is an extremal topology on a set \(X\) then:

a) \((X, \tau)\) is a normal space.

b) \((X, \tau)\) is a door space and hence a \(T_\delta\) space.

**Corollary 5:**
If \(\tau = P(X\{x\}) \cup \{x\} \cup F: F \in \mathcal{F}\) is an extremal topology on \(X\) and \(\mathcal{F}\) is a free ultrafilter in \(X\{x\}\), then \((X, \tau)\) is a \(T_2\) space and hence a \(T_4\) space.

**References**


Electric Quadrupole Moment of Even-Even Nuclei

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Abstract

In the framework of hydrodynamic model, a new calculations of the electric quadrupole moment of even-even nuclei has been obtained using the first excited state energy of rotational band. With the assumption that nuclei are volume preserving under rotation, the radii of spheroid have also been computed. In this work, the isotope of Dy164 showed a noticeable difference in size.

Keywords: Inertia; Charge; Quadrupole; Rotation; Spheroid; Spin.

المستخلص

في إطار النموذج الهيدروديناميكي ، أجريت حسابات جديدة للعزم الكهروي رباعي القطب للانوية الزوجية-الزوجية من خلال توظيف مستوى الأائرة الأول في النطاق الدوراني ، وذلك بفرض أن الأائرة تابعة الحجم أثناء عملية الدوران حيث تم أيضا حساب أطوار أقسام أقطار شبه الكرة . في هذا البحث أظهر نصير Dy164 اختلافا ملحوذا في الحجم.

Introduction

A nucleus with unpaired nucleons will have a charge distribution which results in an electric quadrupole moment. Properties of these nuclei with several nucleons outside a closed shell are described in a first approximation by their interactions with an inert core plus other nucleons which can interact with the core and mutually with each other via a residual interaction [1], [2].

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The allowed nuclear energy levels are shifted unequally due to the interaction of the nuclear charge with an electric field supplied by the non-uniform charge distribution [3]. One of the main causes of nuclear deformation is made apparent by connection with electric quadrupole moments [4], together with the low energy spectrum contains sequences of rotational states varying with quantized angular momentum [5].

**Nuclear Electric Quadrupole Moment**

An even-even nucleus with mass \( M \) in the region \( 150 < A < 190 \) can rotate about an axis at right angles to the axis of symmetry, forming an axially-symmetric rigid rotator with uniform mass distribution [6], these rotations can only be observed in nuclei with non-spherical equilibrium [7], [8]. The energy spectrum of such rotator with moment of inertia \( \mathscr{I} \) and quantized angular momentum \( I \) is given by [9]

\[
E_I = \frac{\hbar^2}{2\mathscr{I}} I(I+1)
\]  

with the moment of inertia is taken to be a classical spheroid of rotation

\[
\mathscr{I} = \frac{1}{5} M (a^2 + c^2)
\]  

where \( a \) denotes the distance along the axis of symmetry and \( c \) is the distance along the axis perpendicular to the axis of symmetry. The quantum analogue of the moment of inertia can be obtained from relation [10]

\[
\mathscr{I} = \frac{\hbar^2}{2} \left( \frac{dE}{dI(I+1)} \right)^{-1}
\]  

By definition, the intrinsic quadrupole moment, \( \int d^3r \rho r^2 (3\cos^2 \theta - 1) \) [11], which is evaluated for the case of an axially-symmetric rigid rotator of total number of charges \( Z \) uniformly distributed

\[
Q_0 = \frac{2}{5} Z (a^2 - c^2). 
\]
Electric Quadrupole Moment of Even-Even Nuclei

For the case of well deformed axially symmetric nuclei, the measured quadrupole moment $Q$ can be related to the intrinsic quadrupole moment [12]

$$Q = Q_0 \frac{3K^2 - I(I+1)}{(2I+3)(I+1)}$$

(5)

where $K$ is the projection of total nuclear spin $I$ onto the axis of symmetry, $K = 0$ for even-even axially symmetric nuclei [13].

Table 1. The units of the ground state energy $E_{2s}$ is given in (keV), spheroid volume $a^2 c$ in ($\text{fm}^3$), and quadrupole moment $Q(E_{2s})$ in (b). The observed values listed in last column with no sign if it was not determined by experiment [14].

<table>
<thead>
<tr>
<th>Nucl</th>
<th>$A$</th>
<th>$Z$</th>
<th>$E_{2s}$</th>
<th>$a^2 c$</th>
<th>$Q_{\text{cal}}$</th>
<th>$Q_{\text{exp}}$</th>
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<tr>
<td>Gd</td>
<td>154</td>
<td>64</td>
<td>123</td>
<td>193</td>
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<td>$-1.82(4)$</td>
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<td>254</td>
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<td>$-1.93(4)$</td>
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<td>158</td>
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<td>271</td>
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<td>$-2.01(4)$</td>
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<tr>
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<td>75</td>
<td>240</td>
<td>193</td>
<td>$-2.08$</td>
<td>$-2.08(4)$</td>
</tr>
<tr>
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<td>87</td>
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<td>$1.89$</td>
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<td>189</td>
<td>217</td>
<td>$-1.61$</td>
<td>$-1.6(3)$</td>
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</table>
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Results

Under the assumption that the deformation is volume preserving i.e., $a^2 \propto e^{-1}$, and employing Eq. (2) the radii of spheroid were evaluated by means of any suitable roots finding algorithm [15]. Hence the quadrupole moment for a number of deformed even-even nuclei can be computed using the measured energy of the first excited state $E_2^+$, corresponding to ground state rotational band $K = 0$. Values so obtained are shown in Table 1.

Conclusion

We conclude that in the framework of the nuclear collective model, the quadrupole moments of a number of permanent deformed nuclei were calculated successfully. The obtained values of electric quadrupole moment show good agreement compared with earlier suggested references. Spheroid radii of deformed nuclei in question also obtained. Under the same constraints, we noticed small stretching in volume of listed nuclei when compared with the size of their constituent nucleons. On the other hand, an exception has been noticed in Dy164 which registered quite large deviation. We believe that the calculations carried out are sensitive, and that the nuclear radius constant may conveniently adjusted. Although the odd behavior of Dy164 is surprising and would immediately raise a question.

References

Electric Quadrupole Moment of Even-Even Nuclei

A Simulation Study to Assess the Performance of Three Normality Tests

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Abstract

In this paper, Anderson-Darling (AD), Lilliefors (LI), and Jarque-Bera (JB) tests were compared for Type I error and for power of the tests. The simulation was run 100,000 times for different situations and for different types of departures from normality. For all different sample sizes and distributions, AD gave the most powerful results, followed by the JB test.

Keywords: Type I error; power of the test; Anderson-Darling; Lilliefors; Jarque-Bera tests.

المستخلص

في هذه الورقة، تم مقارنة اختبار أندرسون-دالنج (AD) وأختبار ليليفورس (LI) وأختبار جاركي-بيرا (JB) بالنسبة لخطأ الدرجة الأولى وقوة الاختبار. تم إجراء عدد 100000 محاكاة لحالات مختلفة وأنواع مختلفة بعيدة عن الشكل الطبيعي. لجميع أحجام العينات والتوزيعات، فقد أعطى اختبار AD نتائج أقوى، يليه اختبار JB.

Introduction

Most statistical tests such as $t$-tests, linear regression analysis and Analysis of Variance (ANOVA) require the normality assumptions. Data can be viewed with graphical methods to roughly assess normality. However, graphical methods do not test if the differences between normal distribution and the sample distribution are significant. Tests used for assessing normality are Chi-square, Kolmogorov-Smirnov, Shapiro-Wilks, Anderson Darling, Lilliefors and Jarque-Bera.

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The last three are the most frequently used tests. In most situations, data deviates from normality. Previous studies did not attempt to determine which testing method gives higher power for different cases of sample sizes and distributions and they also had low simulation runs (Ohta and Arizono, 1989; Lin and Mudholkar, 1980).

The main objective of this paper was to evaluate Anderson-Darling (Anderson and Darling, 1952), Lilliefors (Lilliefors, 1967), and Jarque-Bera (Jarque and Bera, 1987) tests for Type I error rates and for power of the tests.

The main three tests that assess assumption of normality are Anderson-Darling (AD), Lilliefors (LI), and Jarque-Bera (JB).

**Anderson Darling Test**

This test for normality, developed by Anderson and Darling (1952), is a popular normality test based on empirical distribution function (EDF) statistics. The Anderson-Darling test is commonly used to test whether a data sample comes from a normal distribution. Let \( x_{(1)} \leq x_{(2)} \leq \cdots \leq x_{(n)} \) be the order statistics of a random sample \( x_1, x_2, \ldots, x_n \) comes from a distribution with cumulative distribution function \( F(x) \). The Anderson–Darling test statistic (AD) is defined by

\[
AD = \frac{-1}{n} \left[ \sum_{i=1}^{n} (2i - 1) \left( \ln \hat{u}_{(i)} + \ln(1 - \hat{u}_{(n-i+1)}) \right) \right] - n;
\]

where

\[
\hat{u}_{(i)} = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{z(i)} e^{-\frac{t^2}{2}} dt, \quad \hat{z}_{(i)} = \frac{x_{(i)} - \bar{x}}{s};
\]

\[
\bar{x} = \frac{1}{n} \sum_{i=1}^{n} x_i \quad \text{and} \quad s^2 = \frac{1}{n-1} \sum_{i=1}^{n} (x_i - \bar{x})^2
\]

are the sample mean and variance (see Gan and Koehler, 1990).

If the resulting AD statistic is significant, then the null hypotheses \( (H_0) \) that the sample comes from a normally distributed population is rejected.
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**Lilliefors Test**

Lilliefors test (LI) is a modification of the Kolmogorov-Smirnov test (Lilliefors, 1967). It is suitable when the unknown parameters of the null distribution must be estimated from the sample data. The LI statistic is defined by

$$LI = \max \left[ \max_{i \in \mathcal{G} \cup \mathcal{U}} \left( \frac{i}{n} - \hat{u}_{(i)} \right), \max_{i \in \mathcal{G} \cup \mathcal{U}} \left( \hat{u}_{(i)} - \frac{i - 1}{n} \right) \right];$$

where

$$\hat{u}_{(i)} = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{\frac{i}{n}} e^{-\frac{t^2}{2}} dt,$$

$$\hat{z}_{(i)} = \frac{x_{(i)} - \bar{x}}{s};$$

$$\bar{x} = \frac{\sum_{i=1}^{n} x_i}{n}$$ and $$s^2 = \frac{\sum_{i=1}^{n} (x_i - \bar{x})^2}{n - 1}$$ are the sample mean and variance.

This test is available when $n$ is greater than or equal to 3.

**Jarque-Bera Test**

The Jarque–Bera (JB) test is a goodness-of-fit test of whether sample data have the skewness and kurtosis matching a normal distribution. Let $x_1, x_2, \ldots, x_n$ be a random sample. The third standardized sample moment of $x_1, x_2, \ldots, x_n$ is called sample skewness and is denoted by

$$\sqrt{b_1} = g_3 = \frac{1}{n} \sum_{i=1}^{n} (x_i - \bar{x})^3 \left( \frac{1}{n} \sum_{i=1}^{n} (x_i - \bar{x})^2 \right)^{3/2}.$$

The fourth standardized sample moment of $x_1, x_2, \ldots, x_n$ is called sample kurtosis and is denoted by

$$b_2 = g_4 = \frac{1}{n} \sum_{i=1}^{n} (x_i - \bar{x})^4 \left( \frac{1}{n} \sum_{i=1}^{n} (x_i - \bar{x})^2 \right)^{2}.$$
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The test statistic denoted by

$$JB = \frac{n}{6} \left( \frac{(b_2 - 3)^2}{4} + \left( \frac{\sqrt{n}}{n} \right)^2 \right),$$

is called Jarque-Bera statistic or just JB statistic, the corresponding test for normality is called Jarque-Bera test or shortly JB test. The test was defined and treated in Jarque and Bera (1987) and their earlier papers. JB is asymptotically chi-squared distributed with two degrees of freedom because JB is just the sum of squares of two asymptotically independent standardized normals, (see Bowman and Shenton, 1975). The Jarque-Bera test is probably the best known normality test to economists and is often used as a test of the normality of residuals.

**Materials and Methods**

A computer simulation program was used to study Monte Carlo techniques to evaluate the power of Anderson Darling (AD), Lilliefors (LI), and Jarque–Bera (JB) test statistics in testing whether a random sample of $n$ independent observations come from a population with a normal distribution. The null and alternative hypotheses are:

- $H_0$: The distribution is normal
- $H_1$: The distribution is not normal.

Matlab R2014a was used to write the program. Type I error rates and statistical power of Anderson and Darling, Lilliefors, and Jarque–Bera tests were measured for different situations. The level of significance $\alpha = 0.05$ was used to investigate the power of the tests. Samples with various sample sizes were taken from the $N(0,1)$, $t(30)$, $\chi^2(30)$, Gamma(2, 3), $\chi^2(3)$, Beta (2, 5), Wiebull(1.5, 1), and Exp (0.50) distributions (Fig. 1).

Random numbers were generated using generators from Matlab R2014a (functions normrnd, trnd, chi2rnd, gamrnd, wblrnd, exprnd, and betarnd) (Moonjung and Wendy, 2015). Sample sizes were chosen as $n = 7, 8, 9, 10 (5) 95, 100 (50) 450, 500 (250) 2000$ for each distribution. This allowed assessment of the Type I error rates and power of statistical tests under small, moderate and large sample size conditions. In each case 100,000 pairs of data sets were generated. Each pair was then compared by each of the three tests. The populations were standardized because they have different means and variances. When samples were taken from $N(0,1)$ populations, the number of rejected $H_0$ hypotheses was declared as the probability for Type I error. When samples were
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taken from populations with non-normal distributions, the number of rejected \( H_0 \) hypotheses was declared as the test’s power. Accordingly, to compute empirical Type I error rate and test power, the program ran each condition 100,000 times and kept tract of proportion of significant statistics.

![Probability density functions of the eight distributions used in the simulation.](image)

**Fig. 1.** Probability density functions of the eight distributions used in the simulation.

### Results and Discussion

The power of the tests varies with the significance level, \( \alpha \), sample size and alternative distributions. However, only the results of power for \( \alpha = 0.05 \), several sample sizes and selected distributions were presented in this paper due to space limitation. The sample sizes presented in the figures were selected at the point which the power dramatically changed.

Empirical results of 100,000 simulation runs are given in the Appendix. Figure 1 below shows the plot of simulated type I error rates for all three tests against the
standard normal distribution for $\alpha = 0.05$. From figure 1 it can be clearly seen that all three tests had similar type I error rates.

Figure 2 below shows the plot of simulated power for all three tests against the $t$-distribution with 30 degrees of freedom for $\alpha = 0.05$. Referring to figure 1 and figure 2 we note that the number of rejected null hypotheses (power of the tests) were similar to the number of rejected null hypotheses (type I error rate) for standard normal distribution when using AD and LI test for sample size less than 100.

Figure 1. Comparison of simulated type I rates for three normality tests against standard normal distribution (skewness = 0.00, kurtosis = 3.00).

Figure 3 displayes the plot of power for the three tests against $\chi^2(30)$ distribution for 5% significance level. It is clear from Figure 3 that the performance of all three tests is low (less than 0.6) for sample sizes less than 150 but JB test performs better than AD and LI tests. JB and AD reached good power (0.6 or more) for sample size of at least 200 while LI requires sample size of at least 300 to reach good power. LI is the weakest test and requires much larger sample size to achieve comparable power with the other two tests.

Figure 4 represents the change in power for different sample sizes for all three tests against Beta(2, 5) distribution. Again the performance of all three tests is low for small sample sizes but in general AD test performs better than JB and LI tests. AD attained good power (0.6 or more) for sample size of 75 or more while JB and LI tests require sample size of 100 or more in order to attain good power.
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Figure 2. Comparison of simulated power for three normality tests against $t$-distribution with 30 degrees of freedom (skewness = 0.00, kurtosis = 3.23).

Figure 3. Comparison of simulated power for three normality tests against chi-squared distribution with 30 degrees of freedom (skewness = 0.51, kurtosis = 3.40).
Figure 4. Comparison of simulated power for three normality tests against Beta (2, 5) distribution ((skewness = 0.6, kurtosis = 2.88).

Figure 5. Comparison of simulated power for three normality tests against gamma (2, 3) distribution ((skewness = 1.41, kurtosis = 6.00).

Figure 5 demonstrates the plot of power for the three tests against Gamma(2, 3) distribution for $\alpha = 0.05$. It is obvious that the performance of all tests is low for small sample sizes but AD and JB test perform better than LI test. AD and JB
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together attained good power (0.6 or more) for sample size of at least 30 while LI requires sample size of at least 40 to attain good power.

Figure 6 shows the plot of power for the three tests against $\chi^2(3)$ distribution for $\alpha = 0.05$. It is clear that for small sample sizes (15 or less) the performance of all three tests is low (less than 0.6). All three tests attained good power (0.6 or more) for sample size of at least 25. Figure 7 above displays the plot of power for the three tests against Weibull(1.5, 1) distribution for $\alpha = 0.05$ while figure 8 below displays the plot of power for the three tests against Exponential(0.5) distribution for $\alpha = 0.05$. It is clear from these plots that the performance of all tests is low for sample sizes less than 15 but in general AD test perform better than JB and LI tests. AD attained good power (0.6 or more) for sample size of 15 or more while JB and LI tests require sample size of 20 or more to attain good power.

Figure 6. Comparison of simulated power for three normality tests against chi-squared distribution with 3 degrees of freedom ((skewness = 1.63, kurtosis = 7.00)).

Conclusion

The results of 100,000 simulation runs showed that;
1. When the distribution is standard normal, any of the three tests can be used to compare Type I error rates.
Figure 7. Comparison of simulated power for three normality tests against Wiebull (1.5, 1) distribution ((skewness = 2.00, kurtosis = 9.00).

Figure 8. Comparison of simulated power for three normality tests against Exponential (0.5) distribution ((skewness = 2.00, kurtosis = 9.00).

1. Regardless of the distribution and sample size, AD test gave higher power levels than the other two tests.
2. In all situations, LI test achieved smallest power levels.
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3. JB and LI tests performed similar in samples with all distributions except for t(30) distribution.
4. All tests were more powerful when used on data with exponential distribution (0.50) and Weibull distribution (1.5, 1) for sample sizes 30 or more.

References

## Appendix

### Type I error rate and power of tests for different distributions and sample sizes

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A Simulation Study to Assess the Performance of Three Normality Tests

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SUGGESTION TO AUTHORS

The Libyan Journal of Science is an international journal, published biannually by the Faculty of Science, University of Tripoli, Libya. The scope of the Journal is to publish original and novel scientific research papers in all different fields of science, pure and applied. Papers which have not been previously published or currently submitted elsewhere will be considered for publication.

All papers submitted to the Libyan Journal of Science are to be assessed by two independent referees and an adjudicator if necessary. The manuscript should be written in good English or Arabic within single column. Authors are required to write and prepare their manuscripts according to the instructions and specifications listed below. An Arabic translation of the abstract will be provided by the Editorial Board where it is not submitted by the author.

Manuscript: The manuscript should be prepared on Microsoft Word documents, using Times New Roman font, on A4 paper size (21cm x 29.7cm) with the margin settings adjusted to 3.5 cm from left and right and 4.5 cm from top and bottom.

Manuscript contents: Authors should organize the contents of their papers according to the following scheme as closely as possible: (1) title of paper; (2) Name(s) of author(s) and affiliation(s) with email(s); (3) Abstract (Arabic and English) and keywords; (4) Introduction; (5) Procedures, techniques, experiments, materials used or studied, areal description etc.; (6) Computations, analysis and results; (7) Discussion; (8) Conclusion; (9) Acknowledgments (optional); (10) References; and (11) Appendices (if included).

Article title: The title should be concise, informative and aligned at the centre and written in Times New Roman in 14 bold with only the first letter capitalized in all. It should be displaced 2 cm below the top margin.

Names of author and affiliations: A list of all authors should be provided with their first and last names written in full, 1 cm below the article title. Affiliations with emails should follow the list of authors, specifically 0.5 cm below the list, and should be indented in the same way as the title and written in regular Times New Roman font with size 11 point.

Abstract: An abstract should accompany each manuscript. It should be completely self-contained, very informative, not exceeding 300 words and written as a single paragraph in Times New Roman font with size 11. The heading title “Abstract” should be centred, written in boldface with size 12 and placed 1cm below the last line of affiliation and 0.5 cm above its main content. The abstract should be justified in alignment and indented 0.5 cm from the left and right margins.
Keywords: A list of five keywords, at most, separated by semicolons is required. The list is placed 0.5 cm below the abstract and 1 cm above the introduction. It should be written with the same font style and size as the abstract.

Main article text: The article text should be divided into sections and subsections (if necessary), specified with a short descriptive title. The main article text should begin with an informative introduction and end with a short conclusion, summarizing the results obtained and the goals achieved. All titled headings for sections should be centred and subsections left justified and written in boldface with size 12. The text should be justified in alignment and written in regular times new roman font with size 12 point.

Acknowledgments and appendices: The acknowledgment (if included) should come after the conclusion in a short section. Technical details that interrupt the flow of any section may be consigned to an appendix. Appendices should be placed after the references (if included). If there are two or more appendices they should be denoted by Appendix A, Appendix B, etc. Equations in an appendix should be numbered as (A.1), (A.2), etc.

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**Genera and Species**: The scientific names of genera and species are to be written in an italic font. This is in accordance with the International Code of Zoological Nomenclature.

**Geographic names**: The National Atlas should be followed for all the geographic names within Libya.

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المجلة الليبية للعلوم
تعليمات النشر

تنشر المجلة الليبية للعلوم مرتين سنوياً عن طريق كلية العلوم بجامعة طرابلس وهي تهدف لنشر البحوث العلمية الأصلية في جميع أفاق العلوم البحتة والتطبيقية وتقبل المجلة مخطوطات البحوث التي لم يسبق نشرها والتي ليست تحت التقييم للنشر في مكان آخر، تتم عملية المراجعات العلمية (التحكيم) عن طريق مختصين آثرين وحالياً مراجع ثالث في حال اختلافهما. إن من مسئولية الكاتب أن تكون الورق مكتوب بلغة عربية سليمة وتتضمن ملخصاً باللغة الإنجليزية (وإذا حصل الأمر) بلغة الإنجليزية.

نص المخطوط: يتم طباعة المخطوط بخط على ورق A4 (29.7×21 سم) بحيث تكون البواسير 3 سم من اليمين واليسار و4.5 سم من الأعلى والأسفل لنرتقي مع شكل المجلة.

محتويات المخطوط: يطلب من الباحثين اعداد مخطوطاتهم حسب النسق التالي أو قريب منه: (1) عنوان البحث، (2) أسم الباحث أو الباحثين كاملاً وعناوينهم البريدي والالكتروني، (3) المستخلص (عربي وإنجليزي) مع كلمات دالة، (4) المقدمة، (5) الطرق والتقنيات والتجارب المستعملة أو المدروس والنطاق الجغرافي، (6) الخ، (7) الحسابات والتحليل والنتائج، (8) المناقشة، (9) الاستنتاجات، (10) المراجع، و(11) الملاحق (إن وجدت).

الاسماء والعناوين: تدرج الأسماء الكاملة أسفل العنوان بخط Simplified Arabic بحجم 16 داكن وأن يكون تحت أعلى الصفحة باستثناء (2) سم.

المستخلص: يجب أن يكون المستخلص متكاملاً ودالاً على محتوي الورقة ولا يزيد عن 300 كلمة، مكتوبه كفرد واحد بخط Simplified Arabic بحجم 11 داكن في منتصف السطر.
الكلمات الدالة: تكتب بنفس خط المستخلص ولا تزيد عن خمس كلمات وتوضع على مسافة 0.5 سم عن المستخلص مباشرة.

النص: يقسم النص إلى أجزاء وأجزاء فرعية (إذا لزم الأمر) بعناوين واضحة، يبدأ النص بمقدمة (تحتوي على المشاكل والدراسات السابقة وهدف البحث) وينتهي بالاستنتاجات التي تلخص النتائج والأهداف المتحصلة عليها، وتوضع عناوين الأجزاء في المركز العناوين الفرعية من اليمين وتكون جميعها بخط 14 داكن على أن يكون النص محبدا من الجهتين.

الملاحظ: توضع المعلومات الفنية التفصيلية التي قد تعوق أنساب النص في ملاحق عقب قائمة المراجع.

المراجع: إن من مسئولية الباحث التأكد من صحة المراجع ودقتها، توضع قائمة بالمراجع التي تتلم الإشارة إليها في النص وفي حال وجود مراجع لم يشتر إليها سيكون المرحورون بخطيا من القائمة وتتم العودة للمراجع باللغة الإنجليزية Suggestions to Authors للإسفل الموجود في التعليمات باللغة الإنجليزية الانجليزية وتتم الإشارة للمراجع باللغة العربية بنفس النسق.

ملخصة: يتم الرجوع للتعليمات باللغة الإنجليزية فيما لم يتم ذكره هنا.

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