

Genetic Disorders in the United Arab Emirates

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The burgeoning research in medical and molecular genetics in the UAE compels the importance of bringing together diverse expertise and resources within the country as well as in the Arab World to provide a forum to foster and support collaborative research to identify the genetic bases of diseases in the region. Considering this, the Centre for Arab Genomic Studies initiated a pilot project to establish a database to catalogue genetic disorders in the Arab World. The initiative mainly aims at the education of the medical community and dissemination of knowledge and reliable information to at-risk populations as the most efficient ways to control genetic disorders in the Arab World.

In accordance with these objectives, we coordinated the collection of data on genetic disorders in the UAE as a model system that we can apply in other Arab countries in the future. The strategy adapted in data collection included the following phases:

- 1. Retrieval of information on genetic disorders in the UAE from specialized public databases:** In depth knowledge of 16 genetic disorders described in the UAE population could be retrieved.
- 2. Screening and collection of data from internationally published literature:** Of approximately 1,250 scientific articles, about 300 contained information on the occurrence of more than 50 genetic disorders in the UAE. These papers were then reviewed, summarized, and edited to suite the format of the planned database.
- 3. Retrieval of data from locally published journals:** More than 70 relevant scientific papers were collected from the Emirates Medical Journal. Less than 10 papers were obtained from journals published in neighboring countries, mainly Saudi Arabia. This stage proved to be an important step in data collection since many locally published

scientific articles contained broad scientific information and included elements of powerful research perspectives (Tadmouri, 2004).

- 4. Mining hospital records:** In Al-Wasl Hospital in Dubai, patient records covering the period from year 1987 to 2004 were prospectively studied. Most of the records were obtained from the Genetics Unit of the hospital that includes the following laboratories: molecular diagnostics, cytogenetics, and biochemistry. About 50 genetic abnormalities were observed in the studied period, these included various types of metabolic disorders, chromosomal abnormalities, and mutations leading to common genetic disorders such as thalassemias, sickle cell disease, and cystic fibrosis. Detailed information down to the mutation level was recorded following a standard method. Similarly, hospital records from Al-Qassimi and Kuwaiti hospitals in Sharjah were collected for years 1993 to 2004. About 50 genetic abnormalities were observed in the studied period, these included various types of cancers, chromosomal abnormalities, and common genetic disorders such as thalassemias, sickle cell disease, and glucose-6-phosphate dehydrogenase deficiency. Currently, patient records of Mafraq Hospital, Corniche Hospital, and Al-Nahyan Clinic in Abu Dhabi are under investigation. Similarly, data from Tawam and Al-Ain hospitals in the city of Al-Ain are being collected. We expect that a large spectrum of information on a variety of genetic disorders will be obtained. It is important to note that data for patients of various nationalities are collected. However, only those obtained from UAE nationals and other Arab patients appear in the database for genetic disorders in Arabs.

During the different phases of data collection, the names of authors active in scientific publishing were recorded and personal communications were organized to obtain further publications and personal observations on the various genetic disorders in the UAE. Succinctly, the magnitude of genetic disorders and congenital abnormalities that we recorded in the UAE population demonstrates the effectiveness of the model strategy that we followed (Table 2).

Table 2 - Genetic disorders in the Arab population of the UAE.

<i>OMIM #</i>	<i>Name</i>	<i>OMIM #</i>	<i>Name</i>
1. 100100	Abdominal Muscles, Absence of, with Urinary Tract Abnormality and Cryptorchidism	36. 141800	Hemoglobin-Alpha Locus 1
2. 100300	Absence Defect of Limbs, Scalp, and Skull	37. 141900	Hemoglobin-Beta Locus
3. 100800	Achondroplasia	38. 142250	Hemoglobin, Gamma G
4. 101400	Saethre-Chotzen Syndrome	39. 142340	Hernia, Diaphragmatic
5. 106100	Angioedema, Hereditary	40. 142623	Hirschsprung Disease
6. 106150	Angiotensin I	41. 142900	Holt-Oram Syndrome
7. 106180	Angiotensin I-Converting Enzyme	42. 143400	Multicystic Renal Dysplasia, Bilateral
8. 107680	Apolipoprotein A-I	43. 143890	Hypercholesterolemia, Autosomal Dominant
9. 107730	Apolipoprotein B	44. 145500	Essential Hypertension
10. 108110	Arthrogyposis Multiplex Congenita	45. 148900	Klippel-Feil Syndrome
11. 108300	Stickler Syndrome	46. 150800	Leiomyoma, Hereditary Multiple, of Skin
12. 108780	Natriuretic Peptide Precursor	47. 151380	Leukemia, Acute Monocytic
13. 108800	Atrial Septal Defect 1	48. 151390	Leukemia, Acute T-Cell
14. 109800	Bladder Cancer	49. 151400	Leukemia, Chronic Lymphocytic
15. 114000	Caffey Disease	50. 151430	B-Cell CLL/Lymphoma 2
16. 114290	Campomelic dysplasia	51. 156810	Microgastria-Limb Reduction Defects Association
17. 114480	Breast Cancer	52. 160900	Dystrophia Myotonica 1
18. 114500	Colorectal Cancer, Hereditary Nonpolyposis	53. 162200	Neurofibromatosis, Type I
19. 115210	Cardiomyopathy, Familial Restrictive	54. 163950	Noonan Syndrome 1
20. 115470	Cat Eye Syndrome	55. 164750	Omphalocele
21. 117550	Sotos Syndrome	56. 166200	Osteogenesis Imperfecta, Type I
22. 117650	Cerebro-costo-mandibular dysplasia	57. 167000	Suppressor of Tumorigenicity 8
23. 118650	Chondrodysplasia Punctata, Autosomal Dominant	58. 167100	Pachydermoperiostosis
24. 120000	Coarctation of Aorta	59. 167750	Pancreas, Annular
25. 120330	Papillorenal Syndrome	60. 168232	Leukemia, Chronic Myeloid
26. 125853	Diabetes Mellitus, Noninsulin-Dependent	61. 172410	Phospholipase A2, Group 1B
27. 131445	Ependymoma, Familial	62. 173000	Pilonidal Sinus
28. 137750	Glaucoma, Primary Open Angle, Juvenile-Onset, 1	63. 173800	Poland Syndrome
29. 137800	Glioma of Brain, Familial	64. 174100	Polydactyly, Imperforate Anus, and Vertebral Anomalies
30. 139090	Gray Platelet Syndrome	65. 176270	Prader-Willi Syndrome
31. 139130	Guanine Nucleotide-Binding Protein, Beta-3	66. 176670	Hutchinson-Gilford Progeria Syndrome
32. 139250	Familial Dwarfism	67. 176880	Protein S, Alpha
33. 139393	Guillain-Barre Syndrome, Familial	68. 178600	Pulmonary Hypertension, Primary
34. 140300	Hashimoto Thyroiditis	69. 179300	Radioulnar Synostosis
35. 141200	Hematuria	70. 179800	Renal Tubular Acidosis
		71. 179820	Renin
		72. 179850	Reticular Pigmented Anomaly of Flexures
		73. 180849	Rubinstein-Taybi Syndrome

<i>OMIM #</i>	<i>Name</i>	<i>OMIM #</i>	<i>Name</i>
74. 187600	Thanatophoric dysplasia	114. 235400	Hemolytic-Uremic Syndrome
75. 188400	DiGeorge Syndrome	115. 236000	Hodgkin Lymphoma
76. 188550	Thyroid Carcinoma, Papillary	116. 236100	Holoprosencephaly 1, Alobar
77. 189960	Tracheoesophageal Fistula with or without Esophageal Atresia	117. 236200	Homocystinuria
78. 190685	Down Syndrome	118. 241400	Hypoparathyroidism
79. 191830	Potter Syndrome	119. 242650	Primary Ciliary Dyskinesia
80. 193000	Vesicoureteral Reflux	120. 243200	Intracranial Hypertension, Idiopathic
81. 193200	Vitiligo	121. 243400	Isoniazid Inactivation
82. 194070	Wilms Tumor 1	122. 243600	Jejunal Atresia
83. 200700	Chondrodysplasia, Grebe Type	123. 245200	Krabbe Disease
84. 201910	Adrenogenital Syndrome	124. 245570	Landau-Kleffner Syndrome
85. 206500	Anencephaly	125. 245600	Larsen Syndrome, Recessive
86. 208500	Jeune thoracic dysplasia	126. 247200	Miller-Dieker Lissencephaly Syndrome
87. 209880	Autonomic Control, Congenital Failure of	127. 248300	Mal de Maleda
88. 213300	Joubert Syndrome 1	128. 248510	Mannosidosis, Beta A, Lysosomal
89. 215100	Chondrodysplasia Punctata	129. 248600	Maple Syrup Urine Disease
90. 216550	Cohen Syndrome	130. 248950	McDonough Syndrome
91. 217095	Conotruncal Heart Malformations	131. 251170	Mevalonate Kinase
92. 218700	Thyroid Dysgenesis	132. 251260	Nijmegen Breakage Syndrome
93. 219800	Cystinosis, Nephropathic	133. 251450	Desbuquois Syndrome
94. 220200	Dandy-Walker Syndrome	134. 252350	Moymoya Disease 1
95. 223340	Phocomelia rt. Hand	135. 252920	Mucopolysaccharidosis Type IIIB
96. 224900	Ectodermal Dysplasia, Anhidrotic	136. 253200	Mucopolysaccharidosis Type VI
97. 225500	Ellis-van Creveld syndrome	137. 253260	Biotinidase
98. 226700	Epidermolysis Bullosa Letalis	138. 254500	Myeloma, Multiple
99. 226730	Epidermolysis Bullosa with Pyloric Atresia	139. 255800	Schwartz-Jampel Syndrome, Type 1
100. 226900	Epiphyseal Dysplasia, Multiple, 4	140. 256100	Nephronophthisis 1
101. 227260	Facial Ectodermal Dysplasia	141. 256300	Nephrosis 1, Congenital, Finnish Type
102. 227600	Factor X Deficiency	142. 256500	Netherton Syndrome
103. 227650	Fanconi Anemia	143. 256700	Neuroblastoma
104. 228520	Fibrochondrogenesis	144. 256800	Insensitivity to Pain, Congenital, with Anhidrosis
105. 229400	Frontofacionasal Dysostosis	145. 257220	Niemann-Pick Disease, Type C1
106. 230000	Fucosidosis	146. 257920	Oculopalatoskeletal Syndrome
107. 230400	Galactosemia	147. 259420	Osteogenesis Imperfecta, Progressively Deforming, with Normal Sclerae
108. 230500	Gangliosidosis, Generalized GM1, Type I	148. 259700	Osteopetrosis
109. 230800	Gaucher Disease, Type I	149. 259900	Hyperoxaluria, Primary, Type I
110. 230900	Gaucher Disease, Type II	150. 261550	Persistent Mullerian Duct Syndrome, Types I and II
111. 231670	Glutaricacidemia I	151. 261800	Pierre Robin Syndrome
112. 231680	Multiple Acyl-CoA Dehydrogenation Deficiency	152. 263200	Polycystic Kidney Disease, Autosomal Recessive
113. 232300	Glycogen Storage Disease II		

OMIM #	Name
153. 263510	Short-Rib-Polydactyly III
154. 263650	Popliteal Pterygium Syndrome, Lethal Type
155. 264480	Pseudotrisomy 13 Syndrome
156. 264600	Pseudovaginal Perineoscrotal Hypospadias
157. 265430	Pulmonary Hypoplasia
158. 265950	Pyloric Atresia
159. 268310	Robinow Syndrome, Autosomal Recessive
160. 268800	Sandhoff Disease
161. 269000	Phocomelia rt. Tibia/Fibia
162. 269250	Schneckenbecken Dysplasia
163. 269700	Lipodystrophy, Congenital Generalized, Type 2
164. 271665	Spondylometaphyseal Dysplasia, Short Limb-Hand Type
165. 271900	Canavan Disease
166. 272800	Tay-Sachs Disease
167. 274600	Pendred Syndrome
168. 276300	Turcot Syndrome
169. 277300	Spondylocostal Dysostosis, Autosomal Recessive 1
170. 277580	Waardenburg-Shah Syndrome
171. 278700	Xeroderma Pigmentosum, Complementa Group A
172. 300068	Androgen Insensitivity Syndrome
173. 300100	Adrenoleukodystrophy
174. 301500	Fabry Disease
175. 302960	Chondrodysplasia Punctata 2, X-Linked Dominant
176. 303350	Masa Syndrome
177. 305000	Dyskeratosis Congenita, X-Linked
178. 305900	Glucose-6-Phosphate Dehydrogenase
179. 307000	Hydrocephalus due to Congenital Stenosis of Aqueduct of Sylvius
180. 307030	Hyperglycerolemia
181. 308840	L1 Cell Adhesion Molecule
182. 309400	Menkes Disease
183. 309550	Fragile Site Mental Retardation 1 Gene
184. 395800	Glomerulonephritis
185. 600631	Enuresis, Nocturnal 1
186. 600807	Astma Susceptibility
187. 601161	Trisomy 18-Like Syndrome
188. 601170	Muscular Dystrophy, Congenital, with Severe Central Nervous System Atrophy and Absence of Large Myelinated Fibers

OMIM #	Name
189. 601446	Right Pelvic Kidney
190. 601451	Nevo Syndrome
191. 601559	Stuve-Wiedemann Syndrome
192. 602089	Hemangioma, Capillary Infantile
193. 602400	Ichthyosis, Follicular Atrophoderma, Hypotrichosis, and Hypohidrosis
194. 602421	Cystic Fibrosis Transmembrane Conductance Regulator
195. 603671	Acromelic Frontonasal Dysostosis
196. 603681	Otoferlin
197. 603903	Sickle Cell Anemia
198. 604801	Muscular Dystrophy, Congenital, 1B
199. 605027	Lymphoma, Non-Hodgkin, Familial
200. 605802	Zinc Finger Homeobox 1B
201. 605818	Deafness, Autosomal Recessive 27
202. 606054	Propionicacidemia
203. 606119	Secreted Ly6/uPAR-Related Protein 1
204. 606545	Ichthyosis, Lamellar, 5
205. 606812	Fumarase Deficiency
206. 606854	Polymicrogyria, Bilateral Frontoparietal
207. 607088	Spinal Muscular Atrophy, Distal, Autosomal Recessive
208. 607364	Bartter Syndrome, Type 3
209. 607398	Glucocorticoid Deficiency 2
210. 607500	Chromosome 18p Deletion Syndrome
211. 607572	Leprosy, Susceptibility to, 2
212. 608091	Cerebellooculorenal Syndrome 2
213. 608911	Choanal Atresia, Posterior

In accordance to other reports, recessively inherited disorders constitute the overwhelming majority of genetic disorders in the UAE population (*Fig. 12*). One of the most plausible explanations for this observation would be the deep-rooted trend of consanguineous marriages widely practiced among the UAE Arab population. However, as children of consanguineous parents are more likely to enter into consanguineous marriages than children of non-consanguineous parents (Al-Gazali *et al.*, 1997), we expect that subpopulations with different gene frequencies and diseases will form. As a result, attention should be directed towards identifying such families and providing them with genetic counseling and access to appropriate services. In addition, careful and detailed analy-

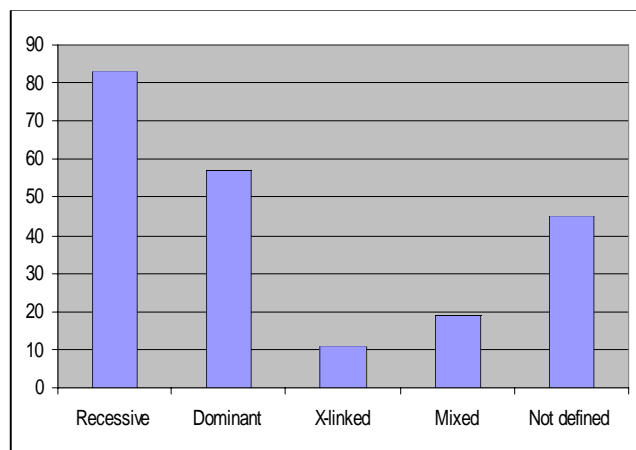


Fig. 12: Modes of inheritance of genetic disorders in the Arab population of the UAE.

sis of many of these anomalies is required in order to offer accurate diagnosis and genetic advice. Besides, these families provide a good opportunity to map and identify the responsible genes in order to improve our knowledge about the mechanisms of normal and pathogenesis of genetic disorders.

At present, we estimate the presence of at least 213 genetic disorders and congenital abnormalities in the Arab population of the UAE. To our knowledge, this is by far the most comprehensive account on this issue in the country. A comparison of the UAE data with the initial information drawn from all Arab populations (Table 1) indicates the presence of 142 new entries. Thus, in total 894 abnormal Mendelian characters do exist in Arabs and this number is expected to rise as we initiate pilot projects to define the correct dimensions of genetic disorders in other Arab countries.

The significance of data collected from the UAE population goes beyond the expansion of our knowledge on the presence of genetic disorders in the country. Various inherited disorders described in the UAE were diagnosed in Arab expatriate groups before they were investigated in their home countries. This makes the data collected so far an important cornerstone in our understanding of the spectrum of genetic disorders in the Arab World.

The Catalogue for Transmission Genetics in Arabs (CTGA)

Data on genetic disorders and congenital abnormalities recorded in the UAE population, including UAE

nationals and non-UAE Arabs, were collected into a database system that we named the "Catalogue of Transmission Genetics in Arabs" (CTGA). As the retrieval of information from the CTGA database is as important as filling data in, we gave a lot of attention to provide the users of the database the option of performing complicated queries to obtain very specific results without sacrificing the simplicity. The current version of CTGA is a textual database whose structure depends on a web-based search that uses an indexing system for rapid retrieval of information. At present, the CTGA database can be queried using two modes of search: basic search or advanced search. In basic search, there is one standard query box in which the user may write one or more keywords. By default, the CTGA search engine automatically processes multiple keywords with the "AND" Boolean operator. The use of wildcards (i.e. the "*" sign that represents any character) is allowed. However, the power of querying at CTGA lies in its advanced search features. At this point, the user can employ a multitude of user-friendly search combinations (e.g. name of disease, disease category, symptoms, gene, OMIM number, chromosome location, mode of inheritance, geographic location, etc.) to increase search sensitivity and to narrow down search results to a small number of positive records (Fig. 13a).

In both types of search, the user issues a command that is interpreted in the CTGA server. Results of the database query are processed by the system's language and are sent to the user's browser as a standard HTML document with no requirement for any additional software. Query results are listed in table form and include the names and corresponding OMIM numbers of genes and genetic disorders described in Arab people. The results page also include helpful components, such as a "search detail" display that reminds the user of the details of the query syntax that was processed, the number of records that matched the search criteria, and the total number of records available in CTGA at the time of the query (Fig. 13b). By selecting a name in the table of results, the user accesses available details related to a specific gene or genetic disorder. Some of the index fields of the detailed record of a genetic disorder includes: the common name and alternative names of the inherited disorder, its category in the World Health Organization International Classification of Diseases, a computer generated map

CTGA
The Catalogue of Transmission Genetics in Arabs

Basic Search

Enter a keyword to search all fields in the database.
To restrict your search, you may use multiple keywords.
There is no need to write 'AND' boolean operator as the CTGA search engine will do this by default.

SEARCH CLEAR

Advanced Search

Enter a keyword or use the selector in the corresponding box to search in a specific field in the database.
To restrict your results, you may use search in multiple fields at a time.
There is no need to write 'AND' boolean operator as the CTGA search engine will do this by default.

Name of Genetic Disease
Symptom, Genetics, Epidemiology
OMIM Number
Reference

Chromosome
 1 2 3 4 5 6 7 8 9 10
 11 12 13 14 15 16 17 18
 19 20 21 22 X Y Not Mapped

Mode of Inheritance
 Autosomal X-linked Y-linked Mitochondrial
 Recessive Dominant

Country
 Algeria Bahrain Egypt Ethiopia Iraq Jordan
 Kuwait Lebanon Libya Mauritania Morocco
 Oman Palestine Qatar Saudi Arabia Sudan
 Syria Tunisia United Arab Emirates Yemen
 Country not specified

SEARCH CLEAR

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a.: Search

CTGA
The Catalogue of Transmission Genetics in Arabs

Viewing records 1 - 10 of 10

Query Summary

Hello localhost
Your search for Gene Locus: 01, Inheritance: recessive, Country: United Arab Emirates, resulted in 10 record(s) of 897 records in the CTGA Database.

Records found

OMIM	Name
106150	Angiotensin I
607364	Barter Syndrome, Type 3
226700	Epidermolysis Bullosa Letalis
230000	Fucosidosis
606812	Fumarase Deficiency
230800	Gaucher Disease, Type I
235400	Hemolytic-Uremic Syndrome
256800	Insensitivity to Pain, Congenital, with Anhidrosis
604801	Muscular Dystrophy, Congenital, 1B
255800	Schwartz-Jampel Syndrome, Type 1

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b.: Results

CTGA
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Name Details →

Synthetic map of the disorder in the Arab World →

Symptoms →

Analyses in Arab people →

References →

Related disorders in CTGA →

External Links for more information →

Record author(s) →

Angiotensin I

Alternative Names
 AGT
 SERPINA8
 Angiotensinogen
 Angiotensin II

OMIM
 106150

Gene Map Locus
 1q42-q43

Mode of Inheritance
 Probably recessive

Description
 Angiotensin is formed from a precursor, angiotensinogen, which is produced by the liver and found in the alpha-globulin fraction of plasma. The lowering of blood pressure is a stimulus to secretion of renin by the kidney into the blood. Renin cleaves from angiotensinogen a terminal decapeptide, angiotensin I. This is further altered by the enzymatic removal of a dipeptide to form angiotensin II.

Molecular Genetics
 The human angiotensinogen molecule has a molecular weight of about 50,000 Da. The angiotensin I decapeptide is located in its N-terminal part. The human angiotensinogen gene is assigned to 1q4 in the same region as the renin gene and contains 5 exons.

Epidemiology in the Arab World
 United Arab Emirates

In 1998, Frossard et al. assessed the value of genotyping the human angiotensinogen gene in a genetically homogeneous population. They carried out a retrospective, case control study of variants M235T and T174M for putative correlations with cardiovascular diseases among UAE. A sample population of 229 Emirati (119 males and 110 females) was investigated. This comprised groups of controls and patients with clinical diagnoses of essential hypertension, left ventricular hypertrophy, ischaemic heart disease and myocardial infarction. M235T and T174M alleles were determined via assays based on the polymerase chain reaction. T174M showed no correlation with any of the four clinical entities included in this study. T235 alleles, however, occurred more frequently in the essential hypertension group and less frequently in the group of myocardial infarction survivors. Frossard and colleagues found that T235 allele frequencies decreased with age, indicating that in the Emirati population, T235 alleles are associated with a reduced life span and that this effect could occur through independent mechanisms underlying genetic susceptibilities to both essential hypertension and myocardial infarction.

References
 > Frossard PM, Hill SH, Elshahat YI, Obineche EN, Bokhari AM, Lestringant GG, John A, Abdulle AM. Associations of angiotensinogen gene mutations with hypertension and myocardial infarction in a gulf population. Clin Genet. 1998; 54(4):285-93.

Related CTGA Records
 > Essential Hypertension
 > Angiotensin I-Converting Enzyme

Links
http://www.orpha.net/consor/cgi-bin/OC_Exp.php?Lng=GB&Expert=112

Contributors
 Ghazi O. Tadmouri: 7.9.2004

Edit History
 Sarah: 10.11.2004
 Ghazi: 28.2.2004

Morocco	Tunisia	Libya	Egypt	Lebanon	Syria	Iraq	Jordan	KSA	Qatar	UAE
Morocco	Algeria	Libya	Egypt	Palestine	Jordan	Kuwait	Oman	Yemen	Bahrain	UAE
Morocco	Algeria	Libya	Egypt	Palestine	Jordan	Kuwait	Oman	Yemen	Bahrain	UAE
Morocco	Algeria	Libya	Egypt	Palestine	Jordan	Kuwait	Oman	Yemen	Bahrain	UAE

c.: Details

Fig. 13: Layouts of the prototype version of the CTGA database: Search (a), Results (b), and Details (c).

showing its geographic occurrence in Arab people, description of the genetic pathology and resulting symptoms, detailed review of research analyses con-

ducted in Arab populations, scientific and general online references, related CTGA records, and others (Fig. 13c).

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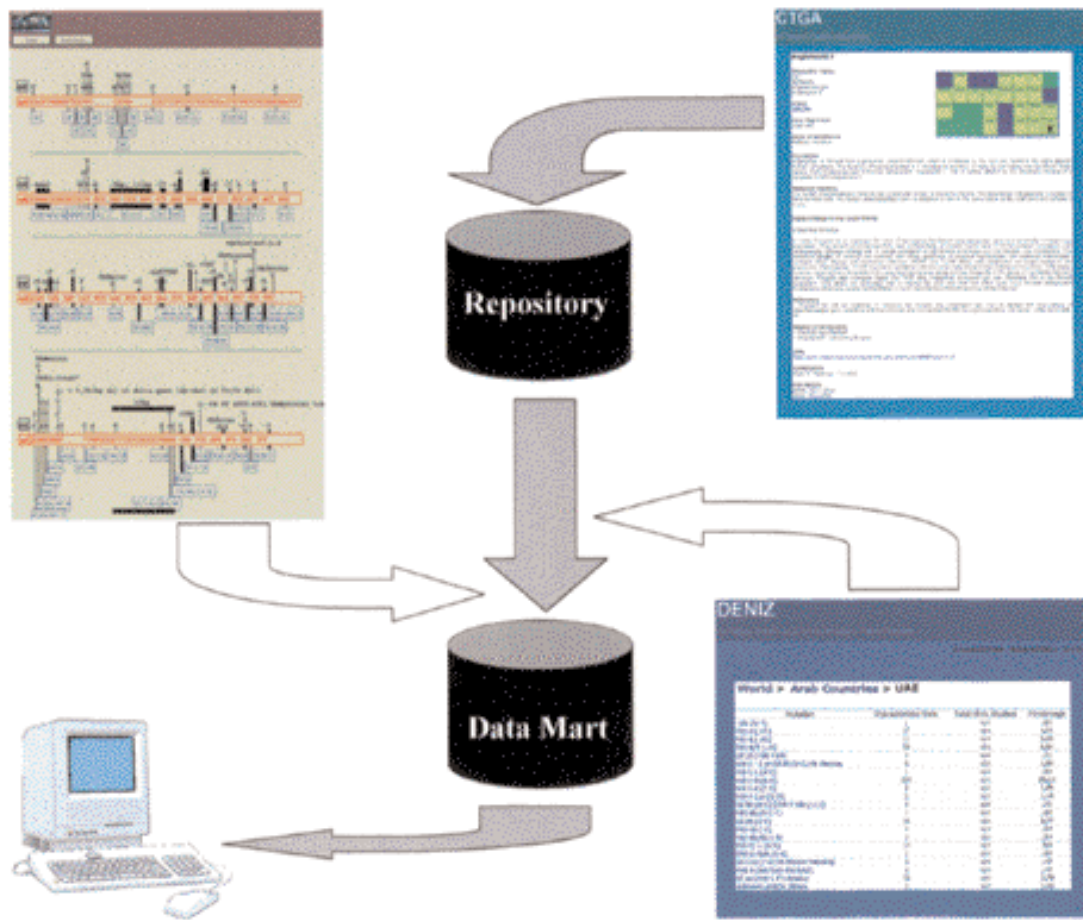


Fig. 14: A schematic presentation of CTGA as a data mart.

At present, the CTGA database is a central information storage facility that consists of a single file composed of records, each of which contains many fields. Although the current priority of CTGA is to publish information on the spectrum of genetic disorders and congenital abnormalities in the UAE, data from other Arab countries are included at this time whenever available.

The language in CTGA is English and the content is directed to researchers and practitioners in the biomedical fields. This is apparent from the detailed clinical description, the overview of molecular pathology, and the extensive review of the literature on the occurrence of each disorder in the Arab World. However, patients and interested individuals can also benefit from the database due to the simple scientific text of each file as well as the presence of various links that point out at a variety of knowledgebase websites

belonging to governmental and non-governmental organizations. Currently, we are adapting the WHO International Classification of Diseases as a standard to categorize genetic disorders in Arabs and to establish a web of links between related records.

Furthermore, CTGA has a flexible structure with high level of experimental detail that can develop in the future into a data mart that collects data from different sources using a relational data model of database management system (DBMS; Fig. 14). For example, advanced users requesting detailed information on the molecular basis of the β -thalassemia disease will have the chance to access resources other than CTGA, such as the DENIZ database, which is the database for world frequencies and biographical information for the various β -thalassemia mutations in world populations (Tadmouri and Gulen, 2003). On the other hand, users with no education in medicine will be able to

access a related database of medical terms so to understand the technical information in CTGA.

To expand the content of CTGA to include detailed data from every Arab country, we will follow a much similar strategy to what we adapted in the case of the UAE. However, data from the UAE will be continuously updated as we monitor, edit, and archive published research. Individual papers will be carefully

reviewed, evaluated, and properly integrated in corresponding records in the CTGA database. Concurrently, the personal contribution of Arab scholars with their data should be an important factor for the continued maintenance and development of the CTGA project. Accordingly, the database will grow as a result of a community effort.
