Mission Statement

The mission of the Neurogenetics Program is to deliver optimal neurological and genetic diagnostic and treatment services to patients with neurological, genetic, or neurogenetic disorders and other developmental disabilities and their families in Lebanon and the region. Also, the program pledges to promote neurological and brain health of children and adults, as well as the legal/social rights of the neurologically impaired and those with dual handicaps. Importantly, the program vows to provide an outstanding academic environment that fosters education, training and state of the art basic research into the causes and potential therapies of pediatric and adult neurological, genetic and neurogenetic disorders. A special mission is application of the multidisciplinary team-centered approach to the care of children with special needs.
1. **MICROARRAY FACILITY**

The microarray facility is fully operational in Diana Tamari Sabbagh Building (DTS) Room 221. The service has a clinical arm and a research arm.

**Clinical Applications:**
(a) Cytogenetics: In cytogenetics chromosomal SNP microarrays or CGH (cytogenetic genomic hybridization) microarrays (eg. Cytogenetics Whole Genome 2.7 M array or CYTO 2.7), DNA is used for analysis of microduplications and/or microdeletions not visible by routine karyotype or FISH.
(b) Copy Number Variation in Cancer. Also, DNA can be used for analysis of copy number variation or CNVs, described in a number of malignancies. For those either CYTO 2.7 or the GeneChip Human Mapping 250K NSP Array can be used.

**Research Applications:**
(c) Genomics Microarray Expression Analysis: Other than the two uses listed above, analysis of RNA expression of experimental samples under different conditions (eg. tumor versus normal tissue, or one tissue versus another or one species versus another may be compared) For this, GeneChip Human Genome 133 Plus 2.0 Array is used.

(a), (b), and (c) are routinely available

(d) *DMET PLUS chips are especially designed for analysis of drug metabolic pathways (more information about these chips are available upon request).
(e) *ChIP-on-Chip microarray is a technique which allows enriching and identifying DNA sequenced bound by regulatory proteins, as well as identifying sites of histone and DNA modification (more information available upon request).
(f) *miRNA Chips: microRNA coverage for 71 organisms allowing research into regulatory stem cell pathways (more information available upon request).

(d), (e) and (f) are available after special consultation
Practical Matters

• All chips will be ordered by the array facility.

• For experimental design, please send an e-mail to Dr. Boustany at rb50@aub.edu.lb or Ext 5640 with a cc. to our technician or call Ext 4806 detailing the exact number and kind of chips you need and the cost center or grant to be billed. It is recommended to briefly meet with Dr. Boustany and outline your experiment.

• Under Affymetrix charges you will find a table with the cost of chips.

• Should you be doing an expression study, you should discuss it with Dr. Claude Chelala (cc17@aub.edu.lb) to plan the experiment (how many samples to run, controls, etc). Otherwise, please feel free to e-mail her and tell her what you need. Alternately, we can have you meet her on future scheduled visits.

• Researchers whose studies require expression analysis by Dr. Chelala will need to budget $2,000/study/year. Dr. Chelala will have confidential computer access to your data from the UK for analysis using PARTEK and other software.
CYTOGENETICS/SNP ARRAYS

Sample Quality and Preparation

I. Background of Cytogenetics Arrays
The Cytogenetics 2.7 M Array gives the greatest power to detect known and novel chromosome aberrations across the entire genome. This array includes 400,000 single nucleotide polymorphisms (SNPs) to enable the detection of loss of heterozygosity (LOH), uniparental disomy (UPD), and regions identical by decent.

II. Genomic DNA Genomic Requirements:
- Starting DNA must be double stranded (not single stranded) for the purpose of the accurate concentration determination.
- DNA must be of high purity. DNA should be free of polymerase inhibitors. DNA purity is indicated by OD260/OD280 and OD260/OD230 ratios. The OD260/OD280 ratio should be between 1.8 and 2.0 and the OD260/OD230 ratio should be greater than 1.5.
- DNA must not be degraded. The appropriate average size of genomic DNA may be assessed on a 1% agarose gel using an appropriate size standard control. Approximately 90% of the DNA must be greater than 10 Kb in size. Control DNA can be run on the same gel for side by side comparison.

III. Sources of Human Genomic DNA:
The following sources of human genomic DNA have been successfully tested in the laboratories at Affymetrix for DNA that meets the requirements.
- Whole Blood (whole blood, white blood cells)
- Cell lines (cultured, fibroblasts, lymphoblasts, fresh tissue, tissue embedded in paraffin)

Other types of samples can be considered depending on the quality (degree of degradation, and the level of purity...) and the quantity of genomic DNA extracted.

IV. Genomic DNA Extraction/Purification Methods:
Genomic DNA extraction is routinely performed using the following methods which have been recommended by Affymetrix:
- SDS/ProK digestion, phenol-chloroform extraction, Microcon or Centricon (Millipore) ultrapurification and concentration.
- Qiagen, QIAmp DNA Blood Kit.
http://www.qiagen.com/products/genomicdnastabilizationpurification/qiaampsystem/qiaampdnabloodmidikit.aspx#Tabs=t2
V. Genomic DNA Preparation/Requirements Needed for the Processing of Cytogenetics Assays:

Human genomic DNA (gDNA) needed for the cytogenetics assay is prepared by:

- Determining the concentration of each sample.
- **Diluting** each gDNA sample to a concentration of **33ng/μl in 1X TE buffer**. Ensure that you start with 100ng gDNA diluted at 33g/μl (100ng total).
- DNA integrity should be reviewed by gel analysis: 1% agarose with ~100 ng DNA. Normalize DNA to 33 ng/ul, then run gel.

![DNA bands in gel](image)

The DNA should appear as a single band with very little smear and the absence of contaminants that are trapped in the loading wells (FFPE=formalin fixed paraffin embedded).

- **gDNA quantity and quality should be**: ≥ 90% of gDNA longer than 10kb by gel; 260/280: 1.8 – 2.0; 260/230: > 1.5; Concentration: 33 ng/ul

**NOTE:**

A copy of the DNA concentrations, absorbance readings of the ratios A260/A280; A260/A230, and an agarose gel picture according to the above criteria should be delivered with the DNA samples. Inadequate concentrations, degraded DNA will fail quality control. One of the steps will fail and the samples and chips would have been wasted. We cannot reimburse anyone for genochips due to bad sample preparation or quality.
RNA Quality and Sample Preparation

I. Background and Overview of 3’ IVT expression Arrays

The GeneChip 3’ IVT Express Kit is the latest technology in RNA target preparation for microarray expression analysis. In the GeneChip 3’ IVT Express Protocol total RNA undergoes reverse transcription to synthesize first-strand cDNA. This cDNA is then converted into a double-stranded DNA template for transcription. In vitro transcription synthesizes aRNA and incorporates a biotin-conjugated nucleotide (cRNA is also known as amplified RNA or aRNA). The aRNA is then purified to remove unincorporated NTPs, salts, enzymes, and inorganic phosphate. Fragmentation of the biotin-labeled aRNA prepares the sample for hybridization onto GeneChip 3’ expression arrays (in this case HG-U133 Plus 2.0). HG-U133 Plus 2.0 Arrays: The Human Genome U 133 Plus 2.0 Array is one microarray comprised of 1,300,000 unique oligonucleotide features covering over 47,000 transcripts and variants, which in turn, represent approximately 39,000 of the best characterized human genes.

II. RNA Extraction/Purification Methods:

RNA Extraction:
The below protocol gives the general steps to extract RNA from tissues, cells, and biological fluids. When working with the RNA reagent (Trizol), work inside the hood and wear protective gloves.

Required Reagents:
- Chloroform
- Isopropanol
- 75% Ethanol
- DEPC treated water

Homogenization:
A- **Tissues**: Homogenize 10-100 mg of fresh tissue with 1 ml RNA reagent in hand held glass Teflon or polytron homogenizer.

B- **Cells**:
- **Cells grown in monolayers**: lyze cells directly in culture dish by adding 1 ml of RNA reagent to a 3.5 cm diameter dish and passing the cell lysate several times through a pipette. The amount of RNA reagent added is based on the area of the culture dish (1ml/10cm2). An insufficient amount of the reagent will result in the contamination of the isolated RNA with DNA.
- **Cells grown in suspension**: Pellet cells by centrifugation then lyse in the RNA reagent by repetitive pipetting (1ml/5-10x10^6 cells).

C- **Biological Fluids**: Use 5 vols of RNA reagent per 1 vol of biological fluid.
RNA Extraction:
- Store the homogenate for 5 minutes at 4°C to permit the complete dissociation of nucleoprotein complexes.
- Add 0.2 ml of chloroform per 1ml of RNA reagent.
- Cover the samples and shake vigorously for 15 seconds.
- Put on ice at 4°C for 5 minutes.
- Centrifuge the homogenate at 12 000 g at 4° C for 15 minutes, the homogenate will form 2 phases the lower phase is the organic phase and the upper phase is the aqueous phase. DNA and protein are in the organic phase and in the interface, while the RNA is in the aqueous phase. The volume of the aqueous phase should be about 40-50% of the total volume of the homogenate plus chloroform.

RNA Precipitation:
- Transfer the aqueous phase to a fresh tube
- Add an equal amount of isopropanol
- Store samples for 10 minutes at 4°C
- Centrifuge at 12 000g at 4°C for 10 minutes.
- RNA precipitation forms a white pellet at the bottom of the tube.

RNA Wash:
- Remove the supernatant
- Wash pellet with 75% EOH (1ml/1ml initial solution used)
- Vortex
- Centrifuge for 5 minutes at 7500g at 4°C
- Repeat the 3 steps above one more time

Re-Dissolving the RNA:
- Remove the supernatant
- Dry the pellet briefly for a couple of minutes (as much ethanol should be removed as possible without completely drying the pellet)
- Dissolve the pellet with DEPC water (volume added depends on the solubility of the pellet but usually 50μl is enough)

Any other possible protocol adapted in your laboratory for RNA extraction could be used but the RNA samples should have the below criteria:

III. RNA Requirements Needed for Processing of the Expression Assays:

*RNA quality is the single most important factor affecting how efficiently an RNA sample will be amplified.*

1. RNA samples should be free of contaminating proteins, DNA, and other cellular material as well as phenol, ethanol, and salts associated with RNA isolation procedures. Impurities can lower the efficiency of reverse transcription and subsequently reduce the level of amplification.
2. An effective measure of RNA purity is the ratio of absorbance readings at 260 and 280 nm. The ratio of A260 to A280 values should fall in the range of 1.7–2.1.

3. The range of the required RNA concentration should be 100-300 ng/µl. RNA must be suspended in sterile, nuclease free, biotechnology grade water.

4. 1% Agarose gel electrophoresis should be used to separate and visualize the major rRNA species. When the RNA resolves into discrete rRNA bands (i.e., no significant smearing below each band), then the mRNA in the sample is likely to be mostly full-length. The below agarose gel picture is an example of how the purified samples should look like (clear band with an unnoticeable smear under).

![Agarose gel picture](image)

**NOTE:**
A copy of the RNA concentrations, absorbance readings of the A260/260 ratio, and an agarose gel picture must be delivered with the RNA samples. Inadequate concentrations, degraded RNA will fail quality control and the samples and chips would have been wasted. We cannot reimburse anyone for wasted Affymetrix genechips due to bad sample preparation or quality.
Affymetrix Charges:

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**Neurogenetics Requisition Form**  (Download Form)

This form should be filled by the physician when referring the patient to the laboratory for the Microarray test.

Kindly click on the link below to access the Neurogenetics Requisition Form

[Neurogenetics Requisition Form.xls]
2. **PROTEOMICS**

Research and Diagnostics Proteomics and Biomarker Research
- Multi-dimensional protein separation
- LC/MS/MS protein identification

**Faculty:**
Firas Kobaissy, PhD
Ext: 4805
Email: [fk02@aub.edu.lb](mailto:fk02@aub.edu.lb)

(Page under Development)
3. RESEARCH

Boustany Research Laboratory:
Our laboratory focuses on uncovering the underlying biochemistry, molecular and cell biology of neuronal storage disorders, in particular, the Neuronal Ceroid Lipofuscinosis, or Batten disease. The hope is uncovering genes, understanding the biochemical and cellular defects, and developing treatments for these progressive and deadly disorders. We have focused mainly on the CLN3, CLN5 and CLN8 genetic variants. Currently, 14 such disorders have been described. The first two abstracts (abstracts 1 and 2) summarize our ongoing work.

One of the genes, CLN3, defective in the juvenile form of NCL, is proving to be a potential biomarker and therapeutic target for cancer, particularly breast cancer (see abstract 3 and 4).

Lastly, our laboratory is interested in uncovering autism susceptibility genes in the Lebanese population (see abstract 5).

1. GALACTOSYLCEERAMIDE (GALCER) AS A POTENTIAL TREATMENT FOR JUVENILE NEURONAL CEROID LIPOFUSCINOSIS (JNCL): JNCL is a neurodegenerative disorder caused by CLN3 gene defects. These negatively modulate cell growth/apoptosis. The CLN3 protein harbors anti-apoptotic motifs and a galactosylceramide (GalCer) lipid raft-binding domain. CLN3p plays a role in GalCer transport to the cell surface, and there is a GalCer deficit in lipid rafts of CLN3-deficient cells. Importantly, CLN3-defective brain/cells have elevated ceramide (Cer). Previous hypotheses suggest that low GalCer in lipid rafts (LR) leads to increased Cer generation in an attempt to overcome the GalCer deficit, and that this leads to neuronal cell death by apoptosis. The $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ knock-in mouse closely mimics the human disorder exhibiting early onset and progressively accumulating JNCL storage material, gliosis, motor disturbances and a shortened life span. Here, to further test this hypothesis, we have examined Cer levels in $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ knock-in mouse brain, and found that they were significantly elevated compared to wild-type littermate control brain Cer levels. We have also tested whether exogenous GalCer administered to homozygous $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mice would correct the reduced LR/Golgi GalCer ratios in homozygous $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mouse brain. Daily intraperitoneal injections of 20 mg/kg GalCer, or vehicle only, were administered to homozygous $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mice between the ages of 5 and 17 weeks. GalCer levels, measured by immunostaining with an anti-GalCer antibody, were increased in thalamus, hippocampus and the cerebellar granular layer in the GalCer-treated versus vehicle-treated $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mice. Exogenous GalCer also decreased the JNCL hallmark storage material levels, measured by subunit c immunostaining, in selected brain regions and in the peripheral tissues examined. In particular, subunit c storage material was dramatically reduced in the liver of GalCer-treated homozygous $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mice versus the vehicle-treated homozygous $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mice. Brains from GalCer-treated homozygous $\text{Cln}^3_{\Delta \text{exc}}^{7/8}$ mice also tended to display reduced gliosis, as measured by GFAP ans S100 immunostaining. In locomotor activity tests, such as pole climbing,
an enhanced benefit for both males and females was observed. It was, however, only statistically significant in males. Finally, addition of GalCer significantly normalized brain ceramide in homozygous Cln3\textsuperscript{Δex7/8} males and females as a group, and this was particularly robust in males. In conclusion, exogenous GalCer partially corrected the GalCer deficit in lipid rafts in tissues from long-term treated homozygous Cln3\textsuperscript{Δex7/8} mice. Moreover, GalCer treatment resulted in diminished subunit c storage in homozygous Cln3\textsuperscript{Δex7/8} tissues, particularly in liver. Significantly, GalCer supplementation also lowered brain ceramide and improved behavior. Thus, our results support the hypothesis that CLN3 defects lead to impaired GalCer trafficking and increased brain ceramide, which may accelerate neurodegeneration in JNCL. GalCer supplementation will be further explored as a treatment option for JNCL.

2. CLN9, CLN5, CLN8 proteins and Ceramide Synthases

Objective: Four patients with a new form of juvenile Neuronal Ceroid Lipofuscinoses (NCL), a childhood neurodegenerative disorder, previously known as CLN9 variant are now reclassified as having CLN5 disease. The various clinical, pathological and biochemical data pertaining to these patients are now attributable to this CLN5 variant. Despite efforts at defining the CLN5 protein, its function remains elusive. CLN5-deficient (CLN5-) fibroblasts obtained from these patients demonstrate adhesion defects, increased growth, apoptosis and decreased levels of ceramide, sphingomyelin, and glycosphingolipids. The CLN8 protein (CLN8p) corrects growth and apoptosis in CLN5-/- cells. This study consolidates the relationship between CLN5 and CLN8 proteins and highlights their role as activators of (dihydro)ceramide synthase (CerS) in a ceramide species-specific manner.

Methods and results: Homozygosity mapping using microarray technology led to identification of CLN5 as the culprit gene. Ceramide synthase activity and ceramide species measured by mass spectrometry are decreased in CLN8-/- cells, similarly to CLN5-/-cells. Comparison of normal vs. CLN5-/- cell CerS1-bound proteins by immunoprecipitation, differential gel Electrophoresis and mass spectrometry, revealed absence of γ-actin in CLN5-/- cells. The γ-actin gene sequence is normal in CLN5-derived DNA. The following γ-actin-bound proteins, vimentin and histone proteins H2Afz/H3F3A/Hist1H4, were absent from the γ-actin protein complex in CLN5-/- cells. Interpretation: The functions of these proteins and their failure to bind to γ-actin could explain the CLN5 cellular phenotype. We explore the role of the CLN5 and CLN8 proteins in sphingolipid de novo biosynthesis and suggest that CLN5 and CLN8 proteins are more closely related than previously believed.

3. TISSUE CLN3 OVEREXPRESSION: A POTENTIAL BIOMARKER FOR BREAST CANCER?

Background: CLN3p is an integral membrane protein encoded by the CLN3 gene. It is anti-apoptotic by decreasing ceramide generation upstream. It is overexpressed at both the mRNA and the protein level in a number of human breast cancer cell lines. We aimed to analyze the clinical significance of CLN3 overexpression in
discrimination of breast tumors (benign vs. DCIS vs. IDC grade I, II and III). **Methods and Results:** RNA is extracted from formalin-fixed paraffin embedded breast tissue blocks, and analyzed by qRT-PCR. 219 samples were analyzed so far (199 DCIS/IDC against 20 benign samples). Receiver-Operator-Characteristic analysis revealed 1.025 as a cut-off value for a fold change that maximizes both sensitivity (77.4%) and specificity (63.9%) and an area under the curve of 0.705 ($P<0.001$).(figure 1).

![CLN3 Expression vs. Benign Breast Tissue](image)

**Figure 1.** ROC analysis of the CLN3 expression quantification in breast cancer (grades I, II and III) vs. DCIS.

**40%** of **DCIS** cases, **73%** of **grade I** IDC, **86%** of **grade II** IDC and **74%** of **grade III** IDC overexpressed **CLN3** (figure 2; $p=0.00004$; chi square).

![CLN3 Overexpression in DCIS and IDC](image)

**Figure 2.** CLN3 overexpression in pre-cancerous and cancerous breast tissue.
Conclusions: The increase in CLN3 overexpression from DCIS (pre-malignant) to IDC (malignant) shows that CLN3p might be a trigger/switch for cancerous transformation of pre-malignant tissue to invasive ductal carcinoma. Quantification of CLN3 expression in breast tissue biopsies may prove to be a novel independent biomarker for differentiating between benign, pre-malignant and malignant tumors. It also may have predictive value related to clinical outcomes.

4. CLN3, a novel molecular target for breast cancer:
Introduction: Breast cancer (BC) is the most common cancer in women, accounting for 23% of all female malignancies. In addition, BC incidence has shown an alarming rate increase worldwide. Elucidation of the underlying biology and molecular pathways for this disease is necessary with the goal of personalizing therapeutic options for individual patients, hence, improving clinical outcomes. The CLN3 protein is anti-apoptotic, and defects in the CLN3 gene cause accelerated apoptotic death of neurons in juvenile Batten disease. Previous work from this laboratory has shown upregulation of CLN3 protein in a number of cancer cell lines, including human and murine breast cancer cell lines, as well as solid colon and breast cancer. Dysregulated apoptotic pathways are often implicated in development of the oncogenic phenotype. The aim of this study is to establish CLN3 expression in a breast cancer cell line (MCF7), and to determine the effect of CLN3 levels on cell growth and apoptosis. Methods: CLN3 expression will be determined by real-time PCR in MCF7 cells compared to normal breast epithelial cells (MCF10A). Blocking CLN3 protein expression will be achieved by transfecting MCF7 cells with scrambled siRNA or with siRNA directed against CLN3. Impact of CLN3 expression on cancer cell growth, apoptosis and ceramide production will be determined using trypan blue dye exclusion, propidium iodide staining, and the DGK assay, respectively. The effect of different chemotherapeutic agents on cancer cell growth and apoptosis will be determined under different CLN3 expression states. Efficacy of Fenretinide, an activator of ceramide generation, sodium butyrate a modifier of histone acetylation, and 5-Aza-2’-deoxycytidine which alters methylation status will be established for different CLN3 expression states. Preliminary results: CLN3 messenger RNA is overexpressed in MCF7 cells compared to MCF10A cells, making MCF7 cells an excellent in vitro model to study the impact of CLN3 expression. We also established the efficacy of CLN3 siRNA showing the blockage of CLN3 protein expression in MCF7 cells. Blocking CLN3 expression inhibited growth and viability of MCF7 cancer cells, and increased apoptosis as shown by propidium iodide staining. Conclusion: Targeting CLN3 overexpression may be an option for therapy in breast cancer. In other words, CLN3 may be a novel molecular target for cancer drug discovery, possibly acting via modulation of ceramide pathways.

5. AUTISM SUSCEPTIBILITY GENES IN THE LEBANESE POPULATION
Introduction: Autism is a neurodevelopmental disorder characterized by three core symptom domains: ritualistic-repetitive behaviors, impaired non-verbal communication and language development. Although autism is considered to be genetic in 20-25% of cases, its extreme heterogeneity has defied genetic classification. Over the last years, the identification of a large number of autism susceptibility genes has led to an increased appreciation of the contribution of de novo and inherited copy number
variation (CNV). Our aims are to confirm previously identified autism susceptibility genes in cases of autism in Lebanon and/or uncover novel autism susceptibility genes specific to the Lebanese population. **Methods:** We are using Cytogenetics 2.7M Microarray technology to detect CNVs in a subset of Lebanese autistic children, and/or to map homozygous regions in each family. The Lebanese population is ideal for homozygosity mapping because of a high degree of shared ancestry and the likelihood of additional healthy siblings per family. **Results:** Homozygous regions specific to autistic children compared to parents and siblings includes previously described autism, schizophrenia and mental disorder susceptibility genes such as CNTNAP2, GLO1, NRXN1 and GRIK2. More interestingly, we uncovered several novel groups of candidate genes. The first subset is involved in glutamatergic transmission already known to be important in autism. These genes are GABRA1, GRIA1 and ALDH9A1. Another subset implicates neuronal cell adhesion molecules including NLGN3, NRXN1 and CNTN3. Other subsets consist of genes like PTPRT and GOLSYN known to play a role in synapse formation, and others related to the mitochondrion and DNA remodeling. Moreover, comparing genomes of 12 autistic children narrowed the analysis to 6 homozygous regions found in 6 or more children. The VPS13B gene associated with Cohen syndrome, a disorder with autistic features was common to 10/12 children. The GPHN gene found in 7/12 patients encodes a protein that plays a role in GABA receptor clustering. **Conclusion:** Uncovering a set of genes responsible for autism in the Lebanese population will facilitate diagnostic work-up and genetic counseling for autistic spectrum disorders in Lebanon. Also, discovery of novel autism susceptibility genes may potentially reveal untapped and novel therapeutic targets.

**Staff**

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Specialties:
- Pediatric Neurology
- Genetics
- Neurogenetics

Medical Conditions:
- All Pediatric Neurologic conditions
- Autism
- Brain Tumors
- Developmental Disorders
- Epilepsy
- Genetic and Neurogenetic Disorders
- Learning Disorders and School-related Problems
- Lysosomal Storage Diseases
- Metabolic Disorders
- Migraine/Headache
- Neurodegenerative Disorders
- Neuromuscular Disorders and Peripheral Neuropathies
- Sleep Disorders
**Faculty**

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<td>Dr. Rose-Mary Boustany</td>
<td>Professor, Head of Pediatric Neurology</td>
<td>1. American Board of Psychiatry and Neurology with Special Qualification in Child Neurology. 2. American Board of Human Genetics</td>
<td>Pediatric Neurology, Genetics</td>
<td><a href="mailto:rb50@aub.edu.lb">rb50@aub.edu.lb</a></td>
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<tr>
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**Educational Programs**

**Residency & Fellowship Program**

The division of Pediatric Neurology offers the opportunity for post-doctoral fellowships that are modeled after the ACGME requirements and approved by the institutional GMEC. The fellowship is three years long and offers training in emergency, inpatient and outpatient pediatric/neurological cases as well as a nine month rotation in adult neurology with electives in epilepsy, neuroradiology, neuropathology, psychiatry or others. There is also the possibility for research in pediatric neuroscience.
The program accepts applicants among AUB graduates and graduates from local or international universities. The choice of the accepted applicants goes through a two-step test. The first step is the English Entrance Exam and the Medical Knowledge Exam (i.e. Dean's Exam for the non-AUB graduates), and the second is the interview with the unit director and/or her/his designee.

The goal of the Pediatric Neurology Fellowship is to provide complete training in Pediatric Neurology for physicians that have completed their training in Pediatrics. The program is modeled according to the requirements and guidelines set jointly by the American Board of Pediatrics and the American Board of Psychiatry and Neurology. In addition to developing expertise in Pediatric Neurology, this program is intended to provide a solid basis in adult Neurology, a requirement in the training of all pediatric neurologists according to the guidelines of the above two boards. It is also intended to provide the trainee with opportunity to perform research and/or to have electives in Neurology subspecialties and related areas.

The training program is modeled after the guidelines of the American Board of Pediatrics and the American Board of Psychiatry and Neurology. These guidelines indicate that trainees should have completed at least two years (can be three) of general pediatrics before starting their pediatric Neurology training. They, then, should have one year of clinical pediatric neurology, 9 months to one year of clinical adult neurology, and a third year that can be spent in further clinical training and/or in any combination of electives in a neurology subspecialty area or in a related field such as neuroscience, neuropathology, or neurosurgery.

In Lebanon, a country of approximately four million inhabitants, there are only ten pediatric neurologists. In addition, in our region of the Middle East there is an even greater need. This is an area with approximately 40 million inhabitants (excluding Egypt). To our knowledge there are only 2 pediatric neurologists in Syria, 4 in Jordan, none in Iraq, eight in Saudi Arabia, and only three in UAE, Qatar, and Bahrain. The patient load at AUB and the expertise available in pediatric and adult neurology as well as in the affiliated fields make the pediatric neurology training program both needed and appropriate.

**Required activities and conferences:**

- Daily morning rounds with the attending physician on the Pediatric Neurology Service.
- EEG reading sessions
- Monday Pediatric Neurology Clinics
- Wednesday morning and afternoon Pediatric Neurology Clinics
- Wednesday noon adult neurology Journal club and case discussion
- Pediatric Neurology Journal Club and Case Discussion
- Friday 8:00-9:00am Pediatric Morbidity Conference
- Monthly epilepsy conference
- Electives:
  a. Neuropathology
  b. EEG (pediatric and adult)
  c. EMG
  d. Neuroradiology
  e. Research electives
  f. Child Psychiatry and Psychology
  g. Epilepsy
  h. Pediatric Ophthalmology – Neuro-ophthalmology

**News on Current/Upcoming Events**
- Lab Meeting Neurogenetics: Friday at 8:30 AM; Weekly
- Journal Club Neurogenetics: Friday at 9:00 AM; Weekly
- Journal Club and Case Discussion Pediatric Neurology, Thursday at 3:00 PM; Bi-weekly

**Residents (2011-2012)**

<table>
<thead>
<tr>
<th>Name</th>
<th>Year</th>
<th>Email</th>
<th>Pager</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dr. Ibrahem Al Shareef</td>
<td>3rd year</td>
<td><a href="mailto:ia25@aub.edu.lb">ia25@aub.edu.lb</a></td>
<td>0318</td>
</tr>
<tr>
<td>Dr. Layal Safadieh</td>
<td>2nd year</td>
<td><a href="mailto:ls40@aub.edu.lb">ls40@aub.edu.lb</a></td>
<td>0283</td>
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<tr>
<td>Dr. Maya Dirani</td>
<td>1st year</td>
<td><a href="mailto:md42@aub.edu.lb">md42@aub.edu.lb</a></td>
<td>0313</td>
</tr>
</tbody>
</table>

**Location**

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Division of Pediatric Neurology  
Neurogenetics Program  
Bldg 23, 1st Floor  
P.O.Box 11-0236  
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Clinic Hours

Dr. Rose-Mary Boustany:
Monday and Wednesday afternoon

Dr. Omar Dabbagh:
Monday, Wednesday and Thursday morning
Tuesday and Friday afternoon

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