Maryland Department of Health & Mental Hygiene

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CRITICAL LINK

June 2008 Volume 12, Number 6

Hemoglobinopathies

Background

Hemoglobin is the iron-containing, multisubunit protein found in red blood cells that transports oxygen from the lungs and transfers it throughout the body. It is a conjugated heme-protein composed of four heme groups and two pairs of polypeptide chains. Differences in peptide number and type of amino acid in the polypeptide chains account for the many types of hemoglobin molecules found in the human population.

In the normal infant at birth, the fetal hemoglobin molecule consists of a pair of alpha and gamma protein chains. After birth, beta chain production replaces the gamma chains to produce the normal adult hemoglobin - a pair of alpha and a pair of beta chains. Mutations in the genes that code for these polypeptides can cause changes in structure and/or function of the hemoglobin molecule and lead to disease. Genetic mutations can result in a variety of hemoglobin dysfunctions such as:

• **Alteration of protein structure**
A mutation of a single subunit (alpha or beta) that does not alter the molecular structure or functionality of hemoglobin, but causes changes in normal hemoglobin function

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Newborn Screening: Recent Politics and Law in Maryland

On April 24, 2008, Governor Martin O'Malley signed House Bill 216-2008[1] into law. This signing signaled a new chapter in the 47-year history of the State of Maryland's public health program to identify treatable hereditary disorders in newborn babies. The new law, entitled "Hereditary and Congenital Disorders—Newborn Screening":

- Designates the Department's Public Health Laboratory as the sole laboratory that may screen the State's newborn population for hereditary disorders;

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Pictured top row, from left: Shawn Cain, OGA, DHMH; Anne Hubbard, OGA, DHMH; Dr. Susan Panny, DHMH; and Mathew Palmer, The Johns Hopkins University. Bottom row, from left: The Hon. Anthony Brown, Lieutenant Governor; The Hon. Mike Miller, Jr., President, Senate; The Hon. Martin O'Malley, Governor; The Hon. Michael Busch, Speaker of the House.

Figure 1: TLIF gels. Hemoglobin samples applied to agarose gels migrate to their isoelectric points and produce focused hemoglobin bands. (Source: Laboratories Administration)

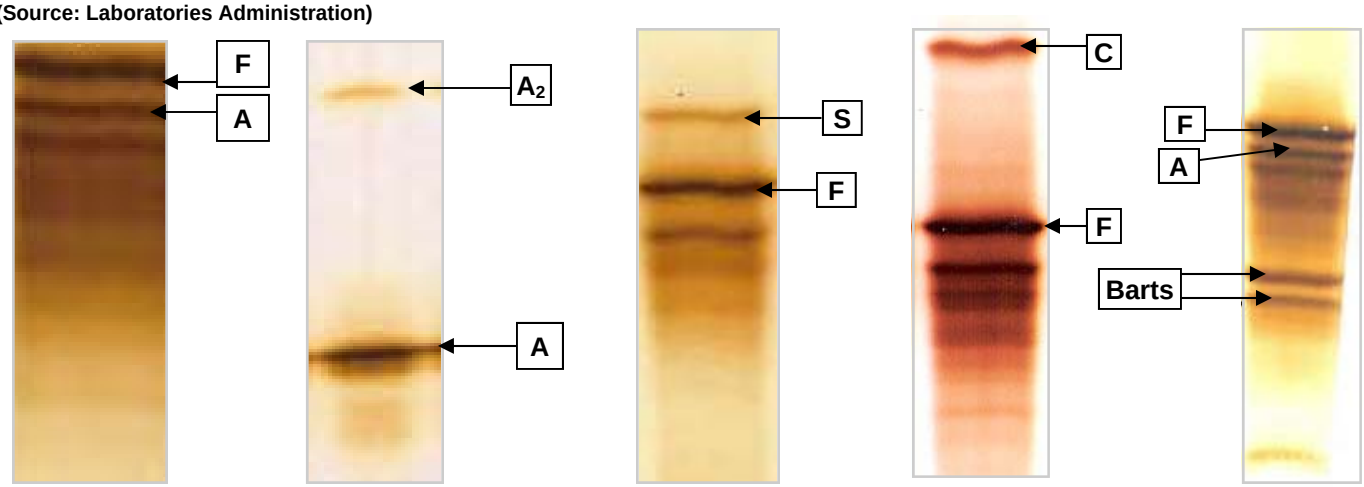

Figure 1a: Normal fetal hemoglobin, note heavy F band at top.

Figure 1b: Normal adult hemoglobin, note predominant A band and lack of F.

Figure 1c: Homozygous S hemoglobin, mutated and indicative of sickle cell disease.

Figure 1d: Homozygous C, also mutated, note the absence of Hemoglobin A in figures 1c and 1d.

Figure 1e: Hemoglobin Barts, indicating gamma chain tetramers and a condition called alpha thalassemia.

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Hemoglobinopathies

producing a diseased state. Sickle cell disease exemplifies this type of dysfunction.[4]

• **Diminished production of hemoglobin subunits**
Normal hemoglobin contains equal amounts of alpha and beta subunits. Diminished production of either subunit produces conditions known as thalassemias[4].

• **Abnormal association of normal subunits**
A single subunit of alpha chain and a single subunit of the beta chain combine to make normal hemoglobin. An example mutation is alpha-thalassemia where 4 beta-globins associate due to scarcity of potential alpha chain partners.[4]

Testing & result analysis

Since 1985, the Department's Hemoglobin Laboratory in the Laboratories Administration has been servicing Maryland citizens with quality and timely results. The Newborn Screening Hemoglobin Laboratory received 13,696 specimens in the first quarter of 2008. Among those, 459 possessed variant hemoglobin traits and 22 were diseased.

The Hemoglobin Laboratory uses two methods to detect abnormal hemoglobins in newborns and adults. The first is thin-layer isoelectric focusing (TLIF), which employs an electrophoretic technique that separates hemoglobins with high resolution. TLIF is based on the migration of hemoglobin proteins to their isoelectric points (pI) when placed under the influence of an electric field. The pH gradient is maintained by a mixture of amphoteric substances that are incorporated in an agarose gel. When hemoglobin samples are applied to the gel, they migrate to their pIs and produce concentrated or focused hemoglobin bands. The relative positions of common hemoglobins from the acidic end (anode) of the gel to the basic end (cathode) are: H, Barts, N, J, A_1C, A, F, S, E/A_2, and C hemoglobins.[3] The advantage of using the isoelectric focusing method to screen for hemoglobins is the ability to isolate different variants present, which helps hematologists identify the location of abnormalities. Maryland's Newborn Screening Division, in collaboration with Isolab modified and optimized the TLIF and has been using this procedure since 1985. A normal hemoglobin pattern is shown in Figure 1a.

The second method employs high performance liquid chromatography (HPLC). This technique separates and determines area percentages for hemoglobins F, A,S,D,C, and E/A_2. As the hemoglobins diffuse within the column, a chromatogram measuring the change in absorbance versus retention time is graphed.[3] The Laboratories Administration scientists use this data to determine genotypes, based on the hemoglobins' relative percents in descending order. HPLC is used as a confirmatory test of hemoglobin disorders. See figure 2.

(Continued on page 3)

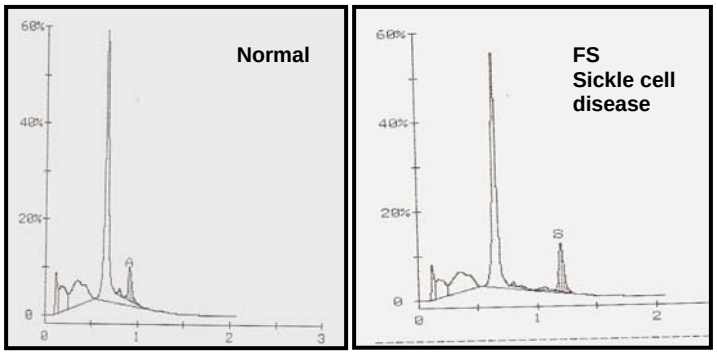

Figure 2: Sample chromatograms, measuring the change in absorbance versus retention time. Source: Laboratories Administration

(Continued from page 2)

Hemoglobinopathies

The common variants reported in various combination by the Laboratories Administration's Hemoglobin Laboratory are:

Hemoglobin F (fetal) (see Figure 1a): The primary oxygen transport protein in the fetus during the last seven months of fetal development is hemoglobin F. Fetal hemoglobin differs most from adult hemoglobin (hemoglobin A), because it is able to bind oxygen with greater affinity than the adult form, allowing the developing fetus better access to oxygen from the mother's bloodstream. In newborns, fetal hemoglobin is nearly completely replaced by adult hemoglobin by approximately the twelfth week of postnatal life.

Hemoglobin A (adult) (see Figure 1b): When fetal hemoglobin production is turned off after birth, normal newborns start producing hemoglobin A. Some patients produce hemoglobin S instead, indicating the presence of sickle cell trait or sickle cell disease.

Hemoglobin S (see Figure 1c): This hemoglobin is predominant in patients with sickle cell disease. The mutation occurs in the beta chain, producing the molecular structure $\alpha_2\beta^S{}_2$. This molecular structure produces sickled (C-shaped) red blood cells which do not allow easy passage of nutrients through the bloodstream. Accumulation of these cells forms clumps and blockages that hamper and impede the proper distribution of oxygen throughout the body. See figure 3.

Hemoglobin C (see Figure 1d): Results from a mutation in the beta globin producing the molecular structure $\alpha_2\beta^C{}_2$. Hemoglobin C disease produces a mild hemolytic anemia, splenomegaly, and earlier than usual breakdown of

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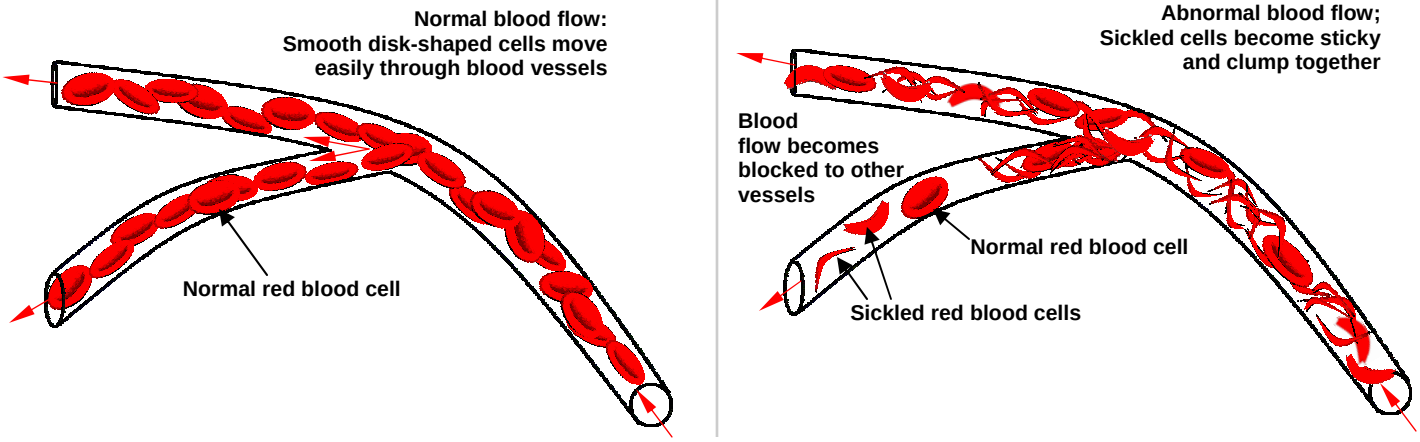

Figure 3: An illustration showing a comparison of normal red blood cells and sickled red blood cells. (Illustration by Georgia Corso, Laboratories Administration, with scientific guidance from Sonja Skeete-Ridley and Diane Young.)

(Continued from page 3)

hemoglobin which changes the plasticity of red blood cells. Hemoglobin C is found mostly in West Africa and has been shown to protect against severe malaria.[7] Patients with this condition are expected to lead normal lives despite the episodes of pain, vision, joint or gallbladder problems they experience.[5]

Hemoglobin Barts (see Figure 1e): A newborn with the presence of hemoglobin Barts at birth indicates a condition called alpha thalassemia (loss of alpha globin genes). Instead of the normal $\alpha_2\beta_2$ formation, the gamma hemoglobins develop in utero and arrange to form gamma chain tetramers. The gamma chains are insoluble in red blood cells and result in poor oxygen delivery to the tissues. The percentage of Barts present in patient blood samples indicates the number of alpha genes lost and the severity of the condition. If the percent is less than 5%, the infant has lost one alpha gene and becomes a normal carrier characteristic of alpha-thalassemia minor. Hemoglobin Barts percentages between 5%-10% indicate the loss of two alpha genes and the presence of alpha-thalassemia minor. If hemoglobin Barts is greater than 10%, a more severe alpha-thalassemia results producing mild anemia with microcytosis (small RBCs). Patients who exhibit hemoglobin Barts at birth typically produce a normal result at their six-month well-baby check. Those who retain hemoglobin Barts at six months may become iron-deficient. Babies determined to be iron-deficient are referred to tertiary centers for care.[2]

Treatments

In 2003, President George W. Bush signed the Sickle Cell Treatment Act into law. This law provides federal funds for patient counseling, educational initiatives and community outreach programs. The law established groundwork for the development of sickle cell treatment centers across the United States to give sickle cell patients the quality healthcare they need.[1]

Maryland has a state-of-the-art program that provides services to its patients with hemoglobinopathies. Once a Laboratories Administration scientist determines an individual may have a diseased condition such as genotype FS/β-thalassemia, the scientist will report the result to the Office for Genetics and Children with Special Health Care Needs (OGCSHCN.) OGCSHCN will follow-up and may request a DNA confirmatory test. The primary care provider is notified so that the patient can receive the appropriate treatment by a team of hematologists, pediatricians, geneticists, and pathologists.

New advances in treatment of patients with hemoglobinopathies have significantly decreased morbidity and mortality in children. Blood transfusions, bone marrow and stem cell transplantation, prophylactic hydroxyurea, and gene therapy have all shown promising signs to improve the quality of life for these patients. Blood transfusions remains the primary treatment sickle cell patients receive to minimize pain.

Bone marrow transplants offer a potential cure for patients with sickle cell disease, but are only used in cases of severe sickle cell disease with minimal organ damage. They require the donor's stem cells to match closely with the recipient's. If there is not a close match, the donor's new immune system may attack certain organs of the recipient causing long-term sickness or death. Bone marrow transplants are successful 85% of the time.[8] The majority of patients who are cured by transplants are children. Adults are unlikely to survive transplants. However, there is a case in Maryland where an adult patient was cured of sickle cell disease, using a donor who was not an exact match. This experimental procedure was developed by a team of doctors at The Johns Hopkins Hospital and is still in the initial stages of clinical trials.[9]

In adults, fetal hemoglobin production can be reactivated pharmacologically. A treatment used for these patients is prophylactic hydroxylurea, a chemotherapeutic agent used in patients with recurring painful vaso-occlusive crises, acute chest syndrome, and other severe sickle cell complications.[6] The agent promotes the production of hemoglobin F, suppresses the blood count of white blood cells and platelets, and inhibits hemoglobin S, reducing the pain a patient experiences.

Gene therapy continues to be explored as a method to inactivate the sickle cell gene and increase the synthesis of hemoglobin F. If successful, gene therapy could provide a permanent correction of the defects in hematopoietic stem cells and lead to new developments that will eventually cure these devastating diseases.

The article was written by Sonja Skeete-Ridley and she would like to acknowledge Diane Young and Carmella Taylor.

References

[1] http://www.sicklecelldisease.org

[2] http://www.scinfo.org/hemoglb.htm

[3] hemoglobin SOPs Laboratories Administration, 2008

[4] http://sickle.bwh.harvard.edu/hemoglobinopathy.html

[5] http://www.nlm.nih.gov/medlineplus/ency/article/000572.htm

[6] http://www.nlm.nih.gov/medlineplus/ency/article/000527.htm

[7] http://en.wikipedia.org/wiki/Hemoglobin_C

[8] http://beehive.thisisnottingham.co.uk/default.asp?WCI=SiteHome&ID=3562&PageID=66242

[9] http://www.baltimoresun.com/news/health/balte.sickle30mar30,0,6112155.story

The scientists of the Hemoglobin Laboratory of the Newborn Screening Program: from left, Diane Young, Newborn Screening Division Chief Dr. Fizza Majid, Sonja Skeet-Ridley, Carmella Taylor, and Maricel Montilla.

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Newborn Screening:
Recent Politics and Law in Maryland

- Calls for a coordinated statewide newborn screening and follow up system;
- Authorizes the Secretary to set fees that, for the first time, include the cost of the follow-up part of the Newborn Screening Program; and
- Allows other laboratories holding a valid Maryland permit to perform second-tier tests that confirm the presence of a disorder.

The State's Newborn Screening (NBS) Program was established in 1965 when the Laboratories Administration first began testing newborns for phenylketonuria (PKU). The Laboratories Administration continued to provide newborn screening for all babies in Maryland, and the Maryland Department of Health and Mental Hygiene (DHMH) supported the belief that only the Laboratories Administration should perform newborn screening on Maryland's babies.

The justification for this was based on the State's responsibility and interest in protecting members of a vulnerable population from suffering the complications (or sequellae) of treatable hereditary disorders. These disorders, if not properly identified and treated, can result in severe and permanent mental retardation and even death, and result in substantial cost to the State in caring for these individuals.

Justification for limiting screening to a single Maryland laboratory was also based on the complexity of the testing, the severe effects of potential laboratory errors, and the need to ensure administrative and follow-up efficiency. Only a single, integrated public health program can ensure reliable communications between the Laboratory and the Follow-Up Unit to quickly identify and track (follow-up) babies that require timely confirmatory testing, access to local treatment centers, and biochemical geneticists.

However, in 2003, a private, out-of-state, newborn screening laboratory claimed that Maryland's newborn screening regulations and law did not limit the performance of newborn screening to the Department's Public Health Laboratory. Faced with the ambiguities of the law, the Secretary at the time ordered that a Maryland permit to perform newborn screening be issued to the private laboratory. Almost immediately after the State permit was issued, the laboratory was sold. The new owners were less familiar with Maryland's NBS Program and continually questioned the State's standards for test performance and reporting of results.

Over the past five years, the existence of two screening laboratories fragmented Maryland's NBS Program. Physicians were no longer sure to which laboratory they needed to send newborn bloodspots. The Department's Public Health Laboratory performed extra testing on specimens that were meant for the private laboratory, rather than risk an untimely or delayed test result. In addition, hospitals using the private laboratory made no

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contribution to help maintain the Follow-Up Unit even though that Unit still provided tracking of all babies found to have abnormal screening results.

However, the greatest risk associated with testing performed by multiple laboratories was the breakdown in communications as the out-of-state lab was not fully integrated into the Department's NBS Program. Thus, results weren't always relayed to the Follow-Up Unit. Results were purposely withheld on certain subpopulations of Maryland newborns (e.g., newborns of military personnel, babies born at home, babies transferred to Children's National Medical Center in Washington, DC).

On a scientific level, each laboratory derived and used slightly different "normal" cutoff points to identify potentially abnormal screening results. The cutoffs were different because each laboratory derived its cutoffs from a different population of newborns. Maryland's State Public Health Laboratory bases its cutoffs on babies born in Maryland. However, the private laboratory bases its cutoffs on babies from across the country, and that laboratory's cutoffs do not reflect as closely the population of babies in Maryland. For example, Maryland's newborn population has a higher percentage of African Americans than many states, which effects the determination of "normal" test result cutoffs. This can lead an out-of-state laboratory to report more false positives that needlessly upset parents and require additional confirmatory testing, or more false negatives that may lead to missing a baby with a treatable hereditary disorder.

Licensing the private laboratory also resulted in a breakdown of oversight by the State's Advisory Council on Hereditary and Congenital Disorders (the Council). For example, when the Council formally recommended that testing of Maryland babies for cystic fibrosis employ Immuno-Reactive Trypsinogen/Immuno-Reactive Trypsinogen (IRT/IRT), the private laboratory insisted on performing Immuno-Reactive Trypsinogen/ Deoxyribonucleic Acid (IRT/DNA). The overall result of using multiple laboratories and related Program inconsistencies put our State's newborns at higher risk. Under such a fragmented system, it was only a matter of time before a baby's results could fall down a communications or scientific crack and the baby suffer irreparable harm.

Beginning in 2004, the Council sought to amend the State's newborn screening law. But it wasn't until the summer of 2007 that the time and circumstances appeared to provide the political will needed to solve this dangerous and highly visible public health problem. At that time, staff from the Family Health Administration (FHA) and the Laboratories Administration worked up an initial draft of House Bill 216-2008. In September 2007, at its monthly meeting, the Council proposed a plan to strongly support passage of the bill with the help of the Secretary and Departmental staff, Council members, representatives of many local and national professional associations, and private individuals. By January 2008, letters of support had been requested and obtained and three panels of supporters had been identified to testify before the appropriate committees in the House and Senate of the General Assembly. In addition, various actions were identified and planned to address the concerns of those who were not in favor of the bill.

Unfortunately, a number of Delegates and Senators mistakenly came to believe that naming the Department's Public Health Laboratory as the sole screening laboratory was an attempt to reverse a move by the previous Governor's

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Maryland's Newborn Screening Program

The Department's NBS Program, consisting of the NBS Laboratory, the Follow-Up Unit, and the Advisory Council on Hereditary and Congenital Disorders, continues to be ranked among the best in the country. Our laboratory and follow-up staff also help maintain our NBS Program's national standing by routinely providing training in NBS to postdoctoral fellows in medical genetics from regional training centers at the National Institutes of Health and the Johns Hopkins Medical Institutions.

Maryland's NBS Program staff also routinely participate in a number of important applied research studies that lead the way nationally to further improvements in newborn screening and an expanded screening menu. One example is an on-going study initiated with Dr. Jennifer Puck, now at the University of California in San Francisco, to develop a new screening test that will identify cases of severe combined immunodeficiency (SCID) in newborns. Others include a continuing project with Dr. Ann Moser at the Kennedy Krieger Institute in Baltimore to develop a newborn screening assay for adrenoleukodystrophy (ALD), and a project with Dr. Joan Marini at NIH on Osteogenesis Imperfecta.

In fiscal year 2007, the Department's NBS Laboratory performed 9.3 million screening assays for more than 50 hereditary disorders (see table of Conditions). Babies tested include newborns from Maryland, newborns of U.S. military dependents in Europe, and occasionally newborns associated with citizens at U.S. embassies overseas.

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Newborn Screening:
Recent Politics and Law in Maryland

administration to privatize newborn screening. This resulted in a more difficult effort to pass what should generally have been seen as an attempt to reduce risk and increase efficiency in a genuine public health program. Following the most recent sale of the private laboratory, its new owners are working closely with the Department to ensure that there is a smooth transition as HB 216-2008 goes into effect.

Recently, the Laboratories Administration sent out a letter to all NBS customers. It informed them of the recent changes that House Bill 216-2008 will bring about, and that the analytical portion of the Program's screening fee per baby would increase from $42 to $60 as of July 1, 2008. This increase is required to pay for actual analytical costs.

The first step in implementing the NBS Program's expansion and reorganization under House Bill 216-2008 is to draft and adopt regulations that set technical standards for newborn screening testing and "coordinate the reporting, follow-up, and treatment activities with parents, caregivers, and health care providers".[1]

The next step is to procure the necessary additional and replacement instrumentation and software required to ensure a continuing NBS program that is reliable and efficient. Software has been under development for several years and is moving towards full implementation. An equipment list has been developed and will be used in preparing a request for a loan from the State Treasurer.

The third step is to prepare job descriptions and recruit additional employees authorized under the bill to fully staff the NBS Program. We expect to begin recruiting this summer for a part-time medical geneticist to serve as Chief of the Follow-Up Unit beginning January 1, 2009. As new staff are recruited and trained for the Follow-Up Unit, they will be located in the Laboratories Administration's Central Laboratory in Baltimore which will further facilitate timely and reliable communications. This will ensure a good and continuing working relationship between the Laboratory and Follow-Up Unit.

Any questions concerning Maryland's NBS Program's laboratory operations may be directed to Dr. Fizza Majid at 410-767-6099, and any questions concerning the Program's Follow-Up Unit may be directed to Dr. Susan Panny at 410-767-6730.

This article written by Drs. Jack DeBoy, Fizza Majid, and Susan Panny.

References

[1] HB 216 DHMH - Hereditary and Congenital Disorders-Newborn Screening, 2008, Chapter 256 [Md. Code Ann. Health General Art. §§ 13-111 and 13-112]
http://mlis.state.md.us/2008rs/chapters_noln/Ch_256_hb0216E.pdf

Table 1: CONDITIONS
2-Methy-3-hydroxy butyric aciduria (2M3HBA)
2-methylbutyryl-CoA dehydrogenase (2MBG)
3MCCC
3-methylglutaconic aciduria tye 1 (3MGA)
Argininaemia
Argininosuccinic aciduria
Biotinidase Deficiency
Carnitine palmitoyltransferase I (CPT 1)
Carnitine palmitoyltransferase II (CPT 2)
Carnitine/acylcarnintine translocase deficiency (translocasae) deficiency (CACT)
Citrullinaemia including:
Citrulinaemia type 1
Citrulinaemia type 2
Congenital Adrenal Hyperplasia (CAH)
CUD, systemic carnitine deficiency, carnitine transporter defect, OCTN2 defect
Cystic Fibrosis
Dienoyl-CoA reductase (De-Red)
Galactosemia including :
Transferase deficient galactosemia
Galactose epimerase
??galactokinase
Glutaric aciduria type 1, GA1
Multiple carboxylase deficiency, (HCS) (MCD)
HMG CoA lyase
Homocystinuria including:
hypermethionininemia
Hypothyroidism including:
Primary hypothyroidism
Secondary hypothyroidism
Transient hypothryoidism
Isobutryl -CoA dehydrogenase (IBCD/IBG) deficiency
Isovaleric acidaemia, IVA
Long-chain hydroxy acyl carnitine dehydrogenase, LCHAD, MTP
MADD, glutaric aciduria type 2, GA2, ETF deficiency
Malonic acidemia (Malonyl-CoA decarboxylase) (MAL)
Maple syrup urine disease, MSUD
MCAD
Methylmalonic acidaemia, MMA, including:
cblA, disease
cblB disease
Mitochondrial acetoacetyl-CoA thiolase (3-ketothiolase) deficiency (BKT)
Phenylketonuria, (PKU) including:
hyperphenylalaninemia
Benign hyperphenylalaninemai
Propionic acidaemia, (PA), ketotic hyperglycinaemia
SCAD
Short chain 3-hydroxy acyl Co-A dehydrogenase (SCHAD) deficiency
Sickle Cell disease including:
Sickle Cell anemia
Sickle Cell C-disease
S-βeta thalasemia
Trifunctional protein (TFP) deficiency
Tyrosinaemia including:
Tyrosinaemia 1
Tyrosinaemia 2
Very long-chain acyl-CoA dehydrogenase (VLCAD)

Laboratory Statistics

NS – Not Speciated
NT – Non-Typeable
VRE – Vancomycin Resistant
SP – Species
NG – No Growth

* This genus has recently been given a new genus name. The genus name in parenthesis is the old name.
** Formerly a part of the *Trichosporon beigelii* complex.
***Alpha streptococci other than *S. pneumoniae* and *Enterococcus*

REPORTED 3/01/08 - 3/31/08

ENTERIC BACTERIOLOGY

GENUS SEROVAR	SEX	AGE	#	JURISDICTION
CAMPYLOBACTER				
	F	42	1	OUT OF STATE
CAMPYLOBACTER JEJUNI				
	U	0	1	ALLEGANY
	F	0	1	ALLEGANY
	M	37	1	BALTIMORE CITY
	F	42	1	OUT OF STATE
	M	0	1	UNKNOWN
ESCHERICHIA COLI				
	F	76	1	BALTIMORE
	M	1	1	BALTIMORE
	M	25	1	BALTIMORE
	U	77	1	BALTIMORE
	M	7	1	MONTGOMERY
	U	0	1	OUT OF STATE
	M	1	1	OUT OF STATE
	U	0	1	OUT OF STATE
SALMONELLA ANATUM				
	M	5	1	FREDERICK
SALMONELLA APAPA				
	M	0	1	BALTIMORE
SALMONELLA DUBLIN				
	M	65	1	PRINCE GEORGE'S
SALMONELLA ENTERITIDIS				
	M	0	1	ALLEGANY
	F	17	1	BALTIMORE
	F	0	1	BALTIMORE
	M	0	1	BALTIMORE
	M	11	1	BALTIMORE
	M	0	1	HARFORD
	F	2	1	MONTGOMERY
	F	48	1	PRINCE GEORGE'S
	M	48	1	BALTIMORE CITY
	F	0	1	BALTIMORE CITY
	F	2	1	BALTIMORE CITY
	F	40	1	BALTIMORE CITY
	F	0	1	BALTIMORE CITY
SALMONELLA HEIDELBERG				
	F	8	1	WASHINGTON
	U	0	1	BALTIMORE CITY
	U	0	1	BALTIMORE CITY
	U	1	1	OUT OF STATE
	U	3	1	OUT OF STATE
SALMONELLA IV 48:G,Z51:-				
	M	0	1	BALTIMORE CITY
SALMONELLA LITCHFIELD				
	F	62	1	OUT OF STATE
SALMONELLA MONS				
	M	0	1	BALTIMORE
	F	3	1	BALTIMORE
SALMONELLA NEWPORT				
	U	75	1	OUT OF STATE
	U	18	1	OUT OF STATE
SALMONELLA SER 4,5,12:I:-				
	M	0	1	WICOMICO
	M	28	1	BALTIMORE CITY
	M	20	1	OUT OF STATE
SALMONELLA SER PARATYPHI B VAR L(+) TARTRATE +				
	M	3	1	BALTIMORE
	M	5	1	BALTIMORE
SALMONELLA STANLEY				
	M	4	1	BALTIMORE CITY
SALMONELLA THOMPSON				
	U	9	1	OUT OF STATE
SALMONELLA TYPHI				
	M	7	1	MONTGOMERY
SALMONELLA TYPHIMURIUM				
	F	4	1	CECIL
	M	12	1	HARFORD
	U	2	1	OUT OF STATE
SALMONELLA TYPHIMURIUM VAR COPENHAGEN				
	F	14	1	BALTIMORE
	M	0	1	MONTGOMERY
	U	3	1	OUT OF STATE
	M	22	1	OUT OF STATE
SHIGELLA FLEXNERI				
	M	25	1	BALTIMORE
	F	91	1	BALTIMORE
SHIGELLA SONNEI				
	U	0	1	ANNE ARUNDEL
	U	4	1	OUT OF STATE
TOTAL			**60**	

ISOLATES – THROAT CULTURES

COUNTY	GROUP A[1]	NON-GROUP A
ALLEGANY	4	19
BALTIMORE	1	0
FREDERICK	1	0
MONTGOMERY	1	1
PRINCE GEORGE'S	1	1
SOMERSET	2	4
WICOMICO	4	8
BALTIMORE CITY	1	3
TOTAL	**15**	**36**

[1] *Streptococcus pyogenes*

BACTERIOLOGY IDENTIFICATIONS
Referrals

GENUS SPECIES	SOURCE	#	JURISDICTION
HAEMOPHILUS INFLUENZAE NON-TYPABLE			
	BLOOD	4	BALTIMORE CITY
	BLOOD	1	WASHINGTON DC
	BLOOD	1	ALLEGANY
	BLOOD	1	PRINCE GEORGE'S
HAEMOPHILUS INFLUENZAE SEROTYPE F			
	BLOOD	1	ANNE ARUNDEL
		1	ST. MARY'S
TOTAL		**9**	

ISOLATES – MISCELLANEOUS

GENUS SPECIES SOURCE	#	JURISDICTION
ACHROMOBACTER SPECIES		
LEG	1	CARROLL
ACINETOBACTER CALCOACETICUS		
FOOT	1	FREDERICK
ACITINOMYCES ODONTOLYTICUS		
BLOOD	1	BALTIMORE CITY
ACITINOMYCES SP.		
BLOOD	1	BALTIMORE CITY
BACILLUS CEREUS		
FOOT	1	FREDERICK
SKIN	1	MONTGOMERY
CLOSTRIDIUM BUTYRICUM		
BLOOD	1	BALTIMORE CITY
ENTEROCOCCUS FAECIUM		
BLOOD	2	BALTIMORE CITY
ESCHERICHIA COLI		
BLOOD	2	BALTIMORE CITY
LUNG	1	BALTIMORE CITY
GARDNERELLA VAGINALIS		
VAGINA	1	CECIL
VAGINA	1	PRINCE GEORGE'S
VAGINA	2	SOMERSET
GEMELLA MORBILORUM		
PERITONEAL	1	BALTIMORE CITY
KLEBSIELLA OXYTOCA		
ABDOMINAL	2	BALTIMORE CITY
KLEBSIELLA PNEUMONIAE		
SPUTUM	1	ANNE ARUNDEL
BLOOD	1	BALTIMORE CITY
PSEUDOMONAS AERUGINOSA		
PERITONEAL	1	BALTIMORE CITY
LEG	1	CARROLL
WOUND	1	FREDERICK
ABSCESS	1	SOMERSET
PSEUDOMONAS PUTIDA		
BLOOD	1	BALTIMORE CITY
OTHER	1	FREDERICK
STAPHYLOCOCCUS AUREUS		
OTHER	1	ALLEGANY
TOE NAIL	1	ALLEGANY
WOUND	1	ALLEGANY
SPUTUM	1	ANNE ARUNDEL
BLOOD	3	BALTIMORE CITY
LUNG	2	BALTIMORE CITY
OTHER	2	BALTIMORE CITY
NASAL	1	CARROLL
WOUND	1	CARROLL
FOOT	3	FREDERICK
OTHER	2	FREDERICK
PENILE LESION	1	FREDERICK
SCROTUM	1	FREDERICK
WOUND	4	FREDERICK
STAPHYLOCOCCUS CAPITIS		
BLOOD	1	BALTIMORE CITY
STAPHYLOCOCCUS COAGULASE NEGATIVE		
BLOOD	1	BALTIMORE CITY
OTHER	1	BALTIMORE CITY
NASAL	2	CARROLL
OTHER	1	CARROLL
FOOT	4	FREDERICK
OTHER	1	FREDERICK
ULCER	1	FREDERICK
ABSCESS	1	MONTGOMERY
STAPHYLOCOCCUS EPIDERMIDIS		
BLOOD	1	BALTIMORE CITY
STREPTOCOCCI ALPHA HEMOLYTIC		
BLOOD	1	BALTIMORE CITY
STREPTOCOCCI BETA HEMOLYTIC GROUP B		
CERVIX	1	ANNE ARUNDEL
VAGINA	1	ANNE ARUNDEL
BLOOD	1	BALTIMORE CITY
VAGINA	1	CECIL
VAGINA	5	HOWARD
VAGINA	2	MONTGOMERY
VAGINA	7	PRINCE GEORGE'S
VAGINA	1	SOMERSET
STREPTOCOCCUS ALPHA-HEMOLYTIC		
FINGER	1	BALTIMORE CITY
WOUND	1	BALTIMORE CITY
STREPTOCOCCUS MITIS		
LUNG	1	BALTIMORE CITY
STREPTOCOCCUS SP.		
BLOOD	3	BALTIMORE CITY
TOTAL	**91**	

SEXUALLY TRANSMITTED DISEASES

GENUS SPECIES SEX	#	JURISDICTION
SYPHILIS SEROLOGY		
M	2	ALLEGANY
F	1	ANNE ARUNDEL
M	4	ANNE ARUNDEL
F	4	BALTIMORE
M	4	BALTIMORE
F	16	BALTIMORE CITY
M	27	BALTIMORE CITY
U	1	BALTIMORE CITY
F	1	CALVERT
M	2	CHARLES
M	1	FREDERICK
M	2	HARFORD
M	1	HOWARD
F	7	MONTGOMERY
M	11	MONTGOMERY
F	16	PRINCE GEORGE'S
M	23	PRINCE GEORGE'S
U	1	PRINCE GEORGE'S
F	1	QUEEN ANNE'S
F	2	TALBOT
M	1	TALBOT
M	1	WASHINGTON
F	5	WICOMICO
M	1	WICOMICO
M	1	WORCESTER
TOTAL	**136**	
CHLAMYDIA TRACHOMATIS		
F	2	ALLEGANY
M	10	ALLEGANY
U	2	ALLEGANY
F	19	ANNE ARUNDEL
M	31	ANNE ARUNDEL
U	2	ANNE ARUNDEL
F	2	BALTIMORE
M	9	BALTIMORE
F	4	BALTIMORE CITY
M	38	BALTIMORE CITY
U	3	BALTIMORE CITY
M	2	CECIL
F	1	FREDERICK
M	2	GARRETT
M	23	HARFORD
F	8	HOWARD
M	2	HOWARD
M	2	KENT
F	12	MONTGOMERY
M	36	MONTGOMERY
F	19	PRINCE GEORGE'S
M	69	PRINCE GEORGE'S
U	1	PRINCE GEORGE'S

	F	1	SOMERSET
	M	9	SOMERSET
	U	1	SOMERSET
	F	4	WASHINGTON
	F	2	WICOMICO
	M	2	WICOMICO
TOTAL		**318**	
NEISSERIA GONORRHEAE			
	F	2	BALTIMORE
	M	1	BALTIMORE
	M	1	CALVERT
	M	1	CAROLINE
	M	1	DORCHESTER
	F	1	HOWARD
	M	1	KENT
	F	2	MONTGOMERY
	M	2	MONTGOMERY
	F	11	PRINCE GEORGE'S
	M	15	PRINCE GEORGE'S
	M	1	QUEEN ANNE'S
	F	1	QUEEN ANNE'S
	M	1	ST. MARY'S
	F	1	TALBOT
	M	4	WICOMICO
	F	1	WICOMICO
	F	1	WORCESTER
	M	2	BALTIMORE CITY
	M	1	OUT OF STATE
TOTAL		**51**	

MYCOBACTERIOLOGY

GENUS SPECIES	SEX	AGE	#	JURISDICTION
MYCOBACTERIUM ABSCESSUS				
	F	48	1	HARFORD
MYCOBACTERIUM AVIUM COMPLEX				
	F	85	1	ANNE ARUNDEL
	F	83	1	ANNE ARUNDEL
	F	29	1	BALTIMORE
	M	68	1	BALTIMORE
	M	0	1	BALTIMORE
	F	47	1	CARROLL
	F	78	1	FREDERICK
	F	81	1	FREDERICK
	M	45	1	FREDERICK
	M	51	1	FREDERICK
	M	72	1	FREDERICK
	F	83	1	FREDERICK
	M	61	1	FREDERICK
	M	69	1	FREDERICK
	F	50	1	FREDERICK
	F	84	1	FREDERICK
	M	70	1	FREDERICK
	M	48	1	HARFORD
	F	83	1	MONTGOMERY
	F	79	1	MONTGOMERY
	F	40	1	MONTGOMERY
	F	61	1	MONTGOMERY
	M	28	1	PRINCE GEORGE'S
	M	79	1	PRINCE GEORGE'S
	M	24	1	PRINCE GEORGE'S
	M	34	1	TALBOT
	F	68	1	WICOMICO
	F	84	1	WICOMICO
	F	82	1	WICOMICO
	M	86	1	WICOMICO
	M	43	1	BALTIMORE CITY
	M	28	1	BALTIMORE CITY
	M	75	1	BALTIMORE CITY
	F	64	1	BALTIMORE CITY
	F	32	1	BALTIMORE CITY
	F	28	1	BALTIMORE CITY
	M	28	1	BALTIMORE CITY
	M	39	1	BALTIMORE CITY
MYCOBACTERIUM CHELONAE				
	F	83	1	MONTGOMERY
	M	32	1	OUT OF STATE
MYCOBACTERIUM FORTUITUM				
	M	32	1	ANNE ARUNDEL
	F	77	1	BALTIMORE
	F	56	1	MONTGOMERY
	F	34	1	MONTGOMERY
	M	35	1	PRINCE GEORGE'S
	F	23	1	PRINCE GEORGE'S
	F	33	1	BALTIMORE CITY
	M	76	1	BALTIMORE CITY
	M	34	1	OUT OF STATE
MYCOBACTERIUM GORDONAE				
	M	0	1	BALTIMORE
	M	38	1	BALTIMORE
	M	63	1	BALTIMORE
	F	38	1	MONTGOMERY
	M	43	1	MONTGOMERY
	M	45	1	MONTGOMERY
	M	79	1	MONTGOMERY
	M	49	1	PRINCE GEORGE'S
	M	51	1	PRINCE GEORGE'S
	M	75	1	BALTIMORE CITY
MYCOBACTERIUM KANSASII				
	M	51	1	MONTGOMERY
MYCOBACTERIUM MARINUM				
	M	47	1	BALTIMORE CITY
	M	56	1	BALTIMORE CITY
MYCOBACTERIUM NEOAURUM				
	M	68	1	MONTGOMERY
MYCOBACTERIUM TUBERCULOSIS				
	M	78	1	BALTIMORE
	M	23	1	HARFORD
	F	17	1	HOWARD
	F	75	1	MONTGOMERY
	M	48	1	MONTGOMERY
	M	70	1	MONTGOMERY
	F	27	1	MONTGOMERY
	M	39	1	MONTGOMERY
	M	85	1	MONTGOMERY
	M	47	1	PRINCE GEORGE'S
	F	17	1	PRINCE GEORGE'S
	M	31	1	PRINCE GEORGE'S
	M	52	1	PRINCE GEORGE'S
	M	25	1	SAINT MARY'S
	M	65	1	SAINT MARY'S
	F	31	1	WICOMICO
	M	62	1	BALTIMORE CITY
	M	55	1	BALTIMORE CITY
	M	25	1	BALTIMORE CITY
MYCOBACTERIUM TUBERCULOSIS COMPLEX				
	M	22	1	ANNE ARUNDEL
	M	66	1	ANNE ARUNDEL
	M	78	1	BALTIMORE
	F	78	1	CALVERT
	M	40	1	CECIL
	M	47	1	FREDERICK

F	17	1	HOWARD
M	48	1	MONTGOMERY
M	56	1	MONTGOMERY
M	70	1	MONTGOMERY
M	72	1	MONTGOMERY
F	27	1	MONTGOMERY
M	28	1	MONTGOMERY
M	58	1	MONTGOMERY
M	32	1	PRINCE GEORGE'S
M	29	1	PRINCE GEORGE'S
F	17	1	PRINCE GEORGE'S
M	31	1	PRINCE GEORGE'S
M	49	1	PRINCE GEORGE'S
M	25	1	SAINT MARY'S
M	65	1	SAINT MARY'S
M	37	1	WICOMICO
M	52	1	WICOMICO
F	61	1	BALTIMORE CITY
M	19	1	BALTIMORE CITY
M	62	1	BALTIMORE CITY
F	61	1	BALTIMORE CITY
M	67	1	OUT OF STATE
M	2	1	OUT OF STATE
MYCOBACTERIUM XENOPI			
F	45	1	BALTIMORE CITY
RAPIDLY GROWING MYCOBACTERIA			
M	28	1	MONTGOMERY
F	83	1	MONTGOMERY
F	40	1	MONTGOMERY
SCOTOCHROMOGENIC MYCOBACTERIA			
M	49	1	WICOMICO
TOTAL		**117**	

MYCOBACTERIUM SUSCEPTIBILITY RESULTS

DURING MARCH, 2008, SUSCEPTIBILITY RESULTS ON
14 ISOLATES OF *M. TUBERCULOSIS* COMPLEX * WERE IDENTIFIED.

TOTAL: 5 DRUG RESISTANT STRAINS FOUND

#	COUNTY	DRUG(S)
1	HOWARD	® to ISONIAZID
1[A]	MONTGOMERY	® to ISONIAZID, RIFAMPIN, RIFABUTIN, PYRAZINAMIDE, and ETHAMBUTOL
1[A]	PRINCE GEORGE'S	® to STREPTOMYCIN, ISONIAZID, RIFAMPIN, RIFABUTIN, and ETHAMBUTOL
1	PRINCE GEORGE'S	® to ISONIAZID and STREPTOMYCIN
1	BALTIMORE CITY	® to ISONIAZID

[A] Meets the case definition of Multi-Drug Resistant Tuberculosis (MDRTB)
® RESISTANT

**Mycobacterium tuberculosis* complex consists of:
M. tuberculosis
M. bovis
M. bovis, BCG
M. africanum
M. microti
M. canettii

MYCOLOGY

GENUS SPECIES	SEX	AGE	#	JURISDICTION
ASPERGILLUS FLAVUS				
	F	60	1	PRINCE GEORGE'S
	F	21	1	TALBOT
ASPERGILLUS FUMIGATUS				
	F	80	1	ANNE ARUNDEL
	U	0	1	ANNE ARUNDEL
	F	0	1	ANNE ARUNDEL
	F	65	1	ANNE ARUNDEL
	M	45	1	CALVERT
	M	57	1	MONTGOMERY
	F	76	1	PRINCE GEORGE'S
	F	95	1	PRINCE GEORGE'S
	F	90	1	TALBOT
	M	78	1	TALBOT
	M	34	1	BALTIMORE CITY
	M	42	1	BALTIMORE CITY
ASPERGILLUS NIDULANS				
	F	59	1	CHARLES
ASPERGILLUS NIGER				
	M	69	1	ANNE ARUNDEL
	M	76	1	BALTIMORE CITY
ASPERGILLUS SPECIES				
	F	81	1	ALLEGANY
ASPERGILLUS VERSICOLOR				
	F	50	1	CHARLES
BIPOLARIS				
	M	34	1	ANNE ARUNDEL
	U	0	1	WICOMICO
CANDIDA ALBICANS				
	F	75	1	ANNE ARUNDEL
	F	24	1	BALTIMORE
	F	26	1	BALTIMORE
	F	66	1	CALVERT
	M	29	1	CECIL
	M	72	1	FREDERICK
	F	10	1	FREDERICK
	F	21	1	HOWARD
	F	50	1	MONTGOMERY
	F	56	1	MONTGOMERY
	F	72	1	MONTGOMERY
	F	81	1	MONTGOMERY
	F	84	1	MONTGOMERY
	M	45	1	MONTGOMERY
	M	60	1	MONTGOMERY
	F	25	1	MONTGOMERY
	F	31	1	MONTGOMERY
	F	32	1	MONTGOMERY
	M	59	1	PRINCE GEORGE'S
	F	85	1	PRINCE GEORGE'S
	F	14	1	PRINCE GEORGE'S
	F	18	1	PRINCE GEORGE'S
	F	29	1	PRINCE GEORGE'S
	F	19	1	PRINCE GEORGE'S
	F	18	1	SOMERSET
	F	19	1	SOMERSET
	F	20	1	SOMERSET
	F	21	1	SOMERSET
	F	22	1	SOMERSET
	F	24	1	SOMERSET
	F	60	1	BALTIMORE CITY
	F	82	1	BALTIMORE CITY
CANDIDA GLABRATA				
	F	24	1	BALTIMORE
	F	91	1	BALTIMORE
	F	65	1	MONTGOMERY
	M	44	1	BALTIMORE CITY

CANDIDA KRUSEI				
	F	38	1	BALTIMORE
	F	60	1	BALTIMORE CITY
CANDIDA PARAPSILOSIS				
	F	19	1	SOMERSET
CANDIDA TROPICALIS				
	M	77	1	CALVERT
COCCIDIOIDES IMMITIS				
	M	38	1	BALTIMORE CITY
	F	72	1	BALTIMORE CITY
CRYPTOCOCCUS NEOFORMANS				
	U	0	1	MONTGOMERY
	F	49	1	PRINCE GEORGE'S
CURVULARIA				
	M	5	1	WICOMICO
	F	53	1	WICOMICO
EXOPHIALA SPECIES				
	M	22	1	MONTGOMERY
FUSARIUM SPECIES				
	F	34	1	FREDERICK
	M	70	1	MONTGOMERY
	M	36	1	PRINCE GEORGE'S
HISTOPLASMA CAPSULATUM				
	M	43	1	BALTIMORE CITY
MICROMONOSPORA SPECIES				
	M	44	1	BALTIMORE
MOULD				
	U	0	1	ANNE ARUNDEL
PAECILOMYCES				
	M	49	1	CHARLES
PENICILLIUM SPECIES				
	F	49	1	CARROLL
	M	41	1	MONTGOMERY
	M	72	1	MONTGOMERY
	F	40	1	PRINCE GEORGE'S
	M	76	1	BALTIMORE CITY
	M	34	1	BALTIMORE CITY
RHIZOPUS ORYZAE				
	F	68	1	ALLEGANY
SACCHAROMYCES CEREVISIAE				
	F	71	1	MONTGOMERY
	F	25	1	PRINCE GEORGE'S
TRICHODERMA				
	F	65	1	MONTGOMERY
TRICHOPHYTON MENTAGROPHYTES				
	F	81	1	ALLEGANY
TRICHOPHYTON RUBRUM				
	F	62	1	ANNE ARUNDEL
TRICHOPHYTON TONSURANS				
	M	13	1	CECIL
	F	3	1	CHARLES
	F	8	1	MONTGOMERY
	F	4	1	TALBOT
	F	8	1	TALBOT
	M	1	1	TALBOT
	M	3	1	TALBOT
	M	9	1	TALBOT
TOTAL			**95**	

WATER MICROBIOLOGY

	# TESTED	# NON-COMPLIANT
COMMUNITY	7	3
NON-COMMUNITY	254	42
TOTAL	**261**	**45**

PARASITOLOGY

GENUS SPECIES	#	JURISDICTION
PROTOZOA		
BLASTOCYSTIS HOMINIS	3	BALTIMORE CITY
	2	HOWARD
	6	MONTGOMERY
	3	PRINCE GEORGE'S
	1	HOWARD
	1	PRINCE GEORGE'S
ENDOLIMAX NANA	2	HOWARD
	10	MONTGOMERY
	5	PRINCE GEORGE'S
ENTAMOEBA COLI	2	HOWARD
	3	MONTGOMERY
	6	PRINCE GEORGE'S
ENTAMOEBA HARTMANNI	1	MONTGOMERY
GIARDIA LAMBLIA	6	HOWARD
	3	PRINCE GEORGE'S
IODAMOEBA BUTSCHLII	1	MONTGOMERY
	3	PRINCE GEORGE'S
TOTAL	**58**	
HELMINTHS		
HOOKWORM	1	HOWARD
TOTAL	**1**	

ARTHROPOD IDENTIFICATION

NONE

TICK IDENTIFICATION

NONE

FOOD SAFETY

FOOD AND SHELLFISH MICROBIOLOGY

	# OF SAMPLES	NOTABLE PATHOGENS
FOOD	82	4 SALMONELLA SP. 9 CAMPYLOBACETER SP.
		# STANDARDS EXCEEDED *
CRABMEAT	0	0
		# STANDARDS EXCEEDED **
SHELLFISH	0	0
SHELLFISH GROWING WATERS	227	
ENVIRONMENTAL SAMPLES	9	
OTHER†		
TOTAL	**318**	**13**

†**CLOSTRIDIUM** BOTULINUM TOXIN / TYPE B INFANT BOTULISM

STANDARDS

* CRABMEAT-FRESH
ESCHERICHIA COLI = LESS THAN 36 MPN/100 GRAM
STANDARD PLATE COUNT = LESS THAN 100,000 PER GRAM

** SHELLFISH
FECAL COLIFORMS = LESS THAN 230 MPN/100 GRAM
STANDARD PLATE COUNT = LESS THAN 500,000 PER GRAM

VIRUS ISOLATION

ISOLATE	SEX	AGE	#	JURISDICTION
ADENOVIRUS				
	M	21	1	PRINCE GEORGE'S
	M	18	1	PRINCE GEORGE'S
	M	18	1	PRINCE GEORGE'S
	M	19	1	PRINCE GEORGE'S
	M	20	1	PRINCE GEORGE'S
	M	18	1	PRINCE GEORGE'S
SUBTOTAL			**6**	
HERPES SIMPLEX VIRUS TYPE 1				
	F	17	1	CARROLL
	F	20	1	CARROLL
	F	21	1	SOMERSET
	F	23	1	BALTIMORE CITY
	F	21	1	BALTIMORE CITY
SUBTOTAL			**5**	
HERPES SIMPLEX VIRUS TYPE 2				
	M	54	1	BALTIMORE CITY
	M	21	1	BALTIMORE CITY
SUBTOTAL			**2**	
INFLUENZA A VIRUS				
	F	93	1	ALLEGANY
	M	20	1	ALLEGANY
	F	18	1	ALLEGANY
	F	77	1	BALTIMORE
	F	0	1	HARFORD
	F	11	1	HARFORD
	M	0	1	HARFORD
	F	29	1	HARFORD
	M	80	1	MONTGOMERY
	F	19	1	PRINCE GEORGE'S
	F	40	1	TALBOT
	M	18	1	TALBOT
	F	70	1	TALBOT
	F	38	1	TALBOT
	M	76	1	WASHINGTON
	F	3	1	BALTIMORE CITY
	M	9	1	BALTIMORE CITY
	M	26	1	BALTIMORE CITY
	M	81	1	BALTIMORE CITY
SUBTOTAL			**19**	
INFLUENZA B VIRUS				
	M	48	1	DORCHESTER
	F	87	1	GARRETT
	F	0	1	HARFORD
	M	1	1	HARFORD
	M	2	1	HARFORD
	M	5	1	HARFORD
	F	9	1	HARFORD
	F	2	1	HARFORD
	M	7	1	HARFORD
	M	8	1	HARFORD
	M	5	1	HARFORD
	M	24	1	HARFORD
	F	33	1	HARFORD
	M	21	1	PRINCE GEORGE'S
	F	50	1	TALBOT
	F	97	1	WASHINGTON
	M	85	1	BALTIMORE CITY
	F	0	1	BALTIMORE CITY
SUBTOTAL			**18**	
PARAINFLUENZA VIRUS 1				
	F	19	1	SOMERSET
SUBTOTAL			**1**	
TOTAL			**51**	

NOTE: PCR (POLYMERASE CHAIN REACTION) TESTING RESULTS CANNOT BE REPORTED THIS MONTH BECAUSE WE ARE TRANSITIONING TO A NEW COMPUTER SYSTEM. WE HOPE TO HAVE PCR STATISTICS BY NEXT MONTH.

VIRAL HEPATITIS

ORGANISM	# OF SPECIMENS	POSITIVES	JURISDICTION
HEPATITIS A			
	1	0	ALLEGANY
	10	0	BALTIMORE
	1	0	CALVERT
	1	0	CARROLL
	1	0	HARFORD
	1	0	HOWARD
	2	0	MONTGOMERY
	2	0	PRINCE GEORGE'S
	3	0	WICOMICO
SUBTOTAL	**22**	**0**	
HEPATITIS B			
	65	1	ALLEGANY
	118	3	ANNE ARUNDEL
	74	0	BALTIMORE
	820	14	BALTIMORE CITY
	7	0	CALVERT
	43	1	CARROLL
	183	0	CECIL
	1	0	CHARLES
	96	0	FREDERICK
	17	0	GARRETT
	73	0	HARFORD
	38	0	HOWARD
	1	0	KENT
	302	8	MONTGOMERY
	438	6	PRINCE GEORGE'S
	3	0	QUEEN ANNE'S
	2	0	SAINT MARY'S
	6	0	SOMERSET
	22	0	TALBOT
	33	0	WASHINGTON
	140	3	WICOMICO
	1	0	WORCESTER
SUBTOTAL	**2,483**	**36**	
HEPATITIS C			
	66	5	ALLEGANY
	133	35	ANNE ARUNDEL
	69	5	BALTIMORE
	279	94	BALTIMORE CITY
	11	0	CALVERT
	46	7	CARROLL
	96	13	CECIL
	2	0	CHARLES
	99	7	FREDERICK
	18	0	GARRETT
	36	1	HARFORD
	4	2	HOWARD
	1	0	KENT
	35	0	MONTGOMERY
	238	4	PRINCE GEORGE'S
	3	0	QUEEN ANNE'S
	2	0	SAINT MARY'S
	2	0	SOMERSET
	22	0	TALBOT
	12	2	WASHINGTON
	42	3	WICOMICO
	1	0	WORCESTER
SUBTOTAL	**1,217**	**178**	
TOTALS	**3,722**	**214**	

RABIES

SOURCE	#	JURISDICTION
CAT	1	BALTIMORE
	1	QUEEN ANNE'S
RACCOON	2	ALLEGANY
	6	BALTIMORE
	1	CAROLINE
	1	CARROLL
	3	CHARLES
	2	FREDERICK
	1	HARFORD
	2	HOWARD
	10	MONTGOMERY
	3	PRINCE GEORGE'S
	2	ST. MARY'S
	1	TALBOT
	1	WICOMICO
	2	WORCESTER
SKUNK	1	FREDERICK
	1	MONTGOMERY
	2	ST. MARY'S
TOTAL POSITIVES	**43**	
TOTAL SPECIMENS	**328**	

CHLAMYDOPHILIA (CHLAMYDIA) PSITTACI

REPORTED QUARTERLY
FOR JANUARY 2008 THROUGH MARCH 2008

NO SPECIMENS RECEIVED

CASE DEF.	LEVEL 1 <14%	LEVEL 2 28%-14%	LEVEL 3 ≥29%	TOTAL
1/2007 -3/2007	407	703	365	**1475**
1/2008 - 3/2008	260	481	283	**1024**

The services and facilities of the Maryland Department of Health and Mental Hygiene (DHMH) are operated on a non-discriminatory basis. This policy prohibits discrimination on the basis of age; ancestry; color; creed; marital status; mental or physical disability; national origin; race; religious affiliation, belief, or opinion; sex; or sexual orientation and applies to the provisions of employment and granting of advantages, privileges and accommodations. The Department, in compliance with the Americans with Disabilities Act, ensures that qualified individuals with disabilities are given an opportunity to participate in and benefit from DHMH services, programs, benefits, and employment opportunities.

NEWBORN & CHILDHOOD SCREENING

STATISTICS FOR MARCH 2008

PRESUMPTIVE POSITIVES

DISORDERS	#
PHENYLKETONURIA	8
MAPLE SYRUP URINE DISEASE	5
HOMOCYSTINURIA	15
TYROSINEMIA	16
ARGININEMIA	6
CITRULLINEMIA	1
GALACTOSEMIA	3
BIOTINIDASE DEFICIENCY	0
HYPOTHYROIDISM	50
HEMOGLOBIN -DISEASE	10
HEMOGLOBIN -BENIGN	378
CONGENITAL ADRENAL HYPERPLASIA (CAH)	30
CYSTIC FIBROSIS	3
FATTY ACID OXIDATIONS	24
ORGANIC ACIDEMIAS	20
ACYLCARNITINE - BORDERLINE	4
ACYLCARNITINE - OTHERS	11

MONTHLY TOTALS

# OF SPECIMENS SCREENED	10,417
NUMBER OF TESTS	1,001,985
% OF UNSATISFACTORY SPECIMENS	2.1

YEAR-TO-DATE CONFIRMED CASES

CONDITIONS	# CONFIRMED
MCAD	2
3MCC	0
SCAD	1
VLCAD	0
GA-I	0
MAPLE SYRUP URINE DISEASE	0
PKU- CLINICALLY SIGNIFICANT VARIANT	1
PKU- NOT CLINICALLY SIGNIFICANT VARIANT	1
GALACTOSEMIA- CLASSICAL GALT DEFICIENCY	0
GALACTOSEMIA - VARIANT	1
BIOTINIDASE DEFICIENCY	0
GALACTOSE EPIMERASE DEFICIENCY	0
PARTIAL BIOTINIDASE DEFICIENCY	0
CAH- CLASSICAL SALT WASTING	0
CAH-NON-CLASSICAL	0
HYPOTHYROIDISM - PRIMARY	4
OTHER HYPOTHYROIDISM	1
SICKLE CELL DISEASE -SS	2
SICKLE CELL DISEASE -SC	1
SICKLE CELL DISEASE -S BETA THALASSEMIA	1
CYSTIC FIBROSIS	1

ENVIRONMENTAL CHEMISTRY

SAMPLES	# NON-COMPLIANT	# TESTED
ASBESTOS		
AIR	0	0
BULK	12	16
AIR QUALITY		
$PM_{2.5}$	0	424
PM_{10}	0	0
RADIATION		
AIR/CHARCOAL FILTERS	0	64
MILK	0	3
WIPES	0	38
RAW WATER	0	11
VEGETATION	0	0
OTHER	0	0
DRINKING WATER		
METALS		
COMMUNITY	17	29
NON-COMMUNITY	4	7
PRIVATE WELLS	72	325
PESTICIDES & PCBs		
COMMUNITY	0	84
NON-COMMUNITY	0	92
PRIVATE WELLS	0	0
VOLATILE ORGANIC COMPOUNDS		
COMMUNITY	2	60
NON-COMMUNITY	0	109
PRIVATE WELLS	0	176
RADIATION		
COMMUNITY	9	41
NON-COMMUNITY	0	0
PRIVATE WELLS	0	10
INORGANICS		
COMMUNITY	0	24
NON-COMMUNITY	4	101
PRIVATE WELLS	4	312
FOOD CHEMISTRY		
SUSPECTED TAMPERING	0	0
MICROSCOPIC FILTH	0	0
LABELING	0	0
SURVEILLANCE	0	3
CHEMICAL CONTAMINATION	0	0
TOTAL	**124**	**1,929**

LEAD ENVIRONMENTAL

TEST	#	ELEV	BRL	UNSAT
TOTAL PAINT	**18**	**17**	**0**	**0**
TOTAL SOIL	**3**	**0**	**3**	**0**
DUST				
FLOOR	297	26	252	0
SILL	504	10	454	0
WELL	196	14	133	0
OTHER	35	7	26	0
TOTAL DUST	**1,032**	**57**	**865**	**0**
GRAND TOTAL	**1,053**	**74**	**868**	**0**

INTERPRETATION OF RESULTS:
# = Number of Samples Received
ELEV= Elevated
BRL= Below Reporting Limit
UNSAT = Unsatisfactory
PAINT Positive in excess of 0.5%
SOIL Action level 400 - 5,000 ppm
DUST Clearance limits: Floor/Other 40 ug/sq ft
Window Sill 250 ug/sq ft
Window Well 400 ug/sq ft

LEAD SCREENING - BLOOD LEAD

CLASS		RANGE ug/dl	# TESTED
MARYLAND			
	I	<10	154
	IIA	14-Oct	9
	IIB	15-19	9
	III	20-44	11
	IV	45-69	0
	V	>69	0
TOTAL			**183**
WASHINGTON DC			
	I	<10	0
	IIA	14-Oct	0
	IIB	15-19	0
	III	20-44	0
	IV	45-69	0
	V	>69	0
TOTAL			**0**

HIV ANTIBODY SCREENING – BLOOD (MARCH 2008)

SPECIMEN SOURCES	TOTAL	POSITIVE EIA	%	POSITIVE WB	%
HEALTH DEPARTMENTS AND CLINICS	2,466	95	3.85%	91	95.79%
HOSPITALS	161	10	6.21%	8	80.00%
DETENTION CENTERS	431	14	3.25%	13	92.86%
PRIVATE PHYSICIANS	14	0	0.00%	0	0.00%
STUDENT HEALTH CLINICS	207	0	0.00%	0	0.00%
EMPLOYEE HEALTH CLINICS	14	0	0.00%	0	0.00%
AUTOPSIES	282	15	5.32%	9	60.00%
ORGAN/TISSUE DONORS	95	2	2.10%	1	50.00%
TOTAL	**3,670**	**136**	**3.71%**	**122**	**89.71%**

VIRAL LOAD SPECIMENS (MARCH 2008)

HIV–1 RNA Copies/ml	$<10^3$	$10^3 – 10^4$	$10^4 – 10^5$	$>10^5$	Totals
ALLEGANY	15	2	0	1	18
MONTGOMERY	98	6	21	8	133
PRINCE GEORGES	55	10	12	9	86
SOMERSET	1	1	0	0	2
WASHINGTON	2	0	2	0	4
WICOMICO	2	0	1	0	3
SUBTOTALS	**173**	**19**	**36**	**18**	**246**
DEPT. OF CORRECTIONS	47	15	29	14	105
TOTALS	**220**	**34**	**65**	**32**	**351**

CRITICAL LINK

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