

Annual Report

Newborn Screening 2007

With Preliminary Data for 2008

October 2009



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Executive Summary

In 2007 there were 85,867 infants born in Washington (an additional 3,077 born at military facilities in our state; military hospitals do not participate in this program). The Department of Health's Newborn Screening Program tested these infants for ten preventable, but potentially deadly or disabling disorders that the Washington State Board of Health has specified in chapter 246-650 Washington Administrative Code (WAC). Among these infants, 70 were affected with a severe form of one of the disorders and were quickly enrolled in appropriate preventive care systems before they suffered irreversible damage from their conditions. In addition, 39 infants were identified with a mild form of one of the disorders that required treatment or close monitoring, and 1,403 infants were identified with abnormalities of hemoglobin that, while not directly harmful, can have important implications for future reproduction choices for the infants and their parents. In these cases the infants' health care providers were notified of the findings, their implications, and were provided a list of resources to help families understand how the findings might impact them.

The department's cost to operate the program, including follow-up, education, and evaluation as well as the laboratory testing, is covered through a fee charged for each infant through the hospital of birth. In 2007 this charge was \$60.90 for each child. This modest investment is typically covered by insurance and other third party payers. In return, the state's health care system realizes significant savings by avoiding the costs of lifetime treatment for disabling conditions.

Introduction

This report is presented in accordance with Washington Administrative Code (WAC) 246-650-040 which calls for an annual report of information on newborn screening to the Board of Health. Information on newborn screening during 2007 and preliminary information on 2008 are presented in the attached series of tables and accompanying explanations. Data relating to all births were extracted from 2007 birth certificates by the department's Center for Health Statistics. These data relate to live-birth occurrences within the state. Data relating to infants detected, infants screened, and costs were extracted from data routinely maintained by the department.

The data exclude information relating to infants born at Madigan Army Medical Center, Oak Harbor Naval Hospital and Bremerton Naval Hospital in 2007. These military hospitals do not participate in Washington's Newborn Screening Program.

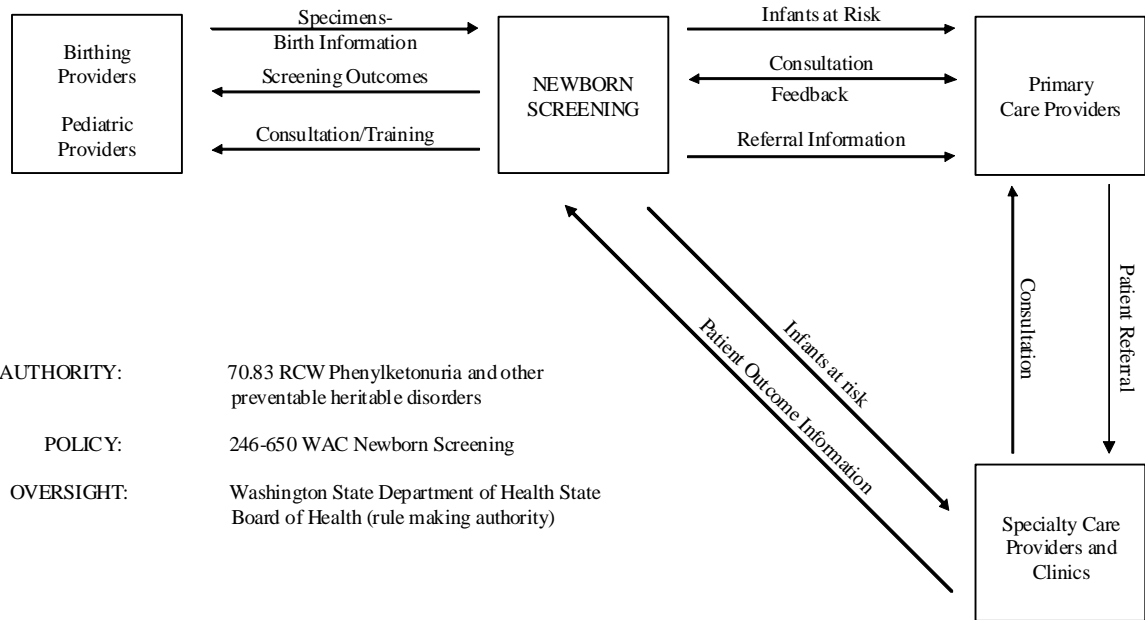
Newborn Screening Schematic Overview

NEWBORN SCREENING

CORE FUNCTION: PREVENTION of severe physical disability or death

METHOD: POPULATION BASED SCREENING of all newborns carefully coordinated with providers of birthing, primary, and specialty care service

FOCUS: PREVENTABLE DISEASE that would go undetected without this screening and result in catastrophic outcomes



The Newborn Screening Program also strives to assure families' involvement in this system through their primary care providers and, for affected infants, through the specialty care providers and clinics.

Description of Disorders and Abbreviations Used

Following is a brief description of the disorders identified through the Newborn Screening Program and abbreviations that are used throughout the report.

- PKU** **Phenylketonuria**; inability to metabolize the common amino acid phenylalanine due to lack of the enzyme phenylalanine hydroxylase. If untreated, PKU results in severe neurological and developmental damage. Treatment consists of a special diet low in phenylalanine. Affected infants develop normally with proper dietary control.
- CH** **Congenital hypothyroidism**; insufficient production of the thyroid hormone thyroxine due to malformation or malfunction of the thyroid gland. If untreated, CH results in severe neurological and developmental damage. Treatment consists of hormone replacement with synthetic thyroxine. Affected infants develop normally with proper treatment.
- CAH** **Congenital adrenal hyperplasia**; excessive production of androgenic hormones due to lack of the enzyme 21-hydroxylase. If untreated, CAH can lead to an imbalance in the body's concentration of salts which in turn can rapidly lead to shock and death. It also causes excessive masculinization and extremely premature sexual maturation. Treatment consists of providing cortisol which normalizes hormone production. Proper treatment prevents death and stops the masculinization process. Affected females may require surgical correction of masculinized genitalia.
- Hb** **Hemoglobinopathies:**
- SCD** **Sickle cell disease**; a condition marked by a tendency for the blood cells to take on a sickle shape due to an abnormal structure of the hemoglobin molecule. The altered shape results in anemia due to shortened life span of the blood cells and impedes circulation, especially in capillaries. Children with sickle cell disease are highly susceptible to bacterial infections that can rapidly lead to overwhelming sepsis and death. Affected children are also vulnerable to rapid pooling of blood in their spleens (splenic sequestration) which can lead to death. Treatment consists of regular doses of penicillin to prevent infection and training parents to recognize splenic crisis. Proper treatment dramatically reduces infections and death.
- Other** **Significant hemoglobinopathies**; hemoglobin abnormalities, other than sickle cell disease, that have significant clinical consequences (for example, transfusion dependent thalassemia). These conditions generally don't require immediate treatment but early identification allows families to adjust to the diagnosis, anticipate the medical needs, and begin early treatment plans as necessary.

Description of Disorders and Abbreviations Used, continued

- GAL** **Galactosemia;** deficiency in enzymes that help convert galactose into glucose. If untreated, jaundice, vomiting, lethargy, hepatosplenomegaly, cataracts and failure to thrive result which can lead to liver failure, sepsis and death. A galactose-free diet with strict avoidance of lactose (milk sugar) and lactose containing foods prevents death, and facilitates normal growth and development.
- BIO** **Biotinidase deficiency;** deficiency of the enzyme that affects normal recycling of biotin, one of the B vitamins. If untreated, a severe deficiency of biotin results leading to metabolic crisis, coma and death. Treatment with unbound biotin can prevent all symptoms.
- MCAD** **Medium Chain Acyl-coA Dehydrogenase deficiency;** deficiency in production of the MCAD enzyme which functions in metabolizing fatty acids. Left untreated there is variable expression, however, fasting or infection can trigger acute episodes of hypoglycemia leading to rapid crisis or death. Treatment consisting of avoidance of fasting, special care during illness, and reduction of dietary fats can prevent all the problems associated with the disorder.
- MSUD** **Maple Syrup Urine Disease;** deficiency of an enzyme needed to break down the branched chain amino acids: leucine, isoleucine and valine. This results in increased serum levels of these amino acids and ketoacid intermediates. Left untreated, death or irreversible brain damage can occur. Treatment consists of a special diet low in the three branched chain amino acids which facilitates normal growth and development.
- HCY** **Homocystinuria;** deficiency of an enzyme necessary for the breakdown of the amino acid methionine. This results in the build up of methionine in the blood and elevated excretion of homocystine in the urine. Left untreated, can result in life-threatening blood clotting, and physical and mental disabilities. Treatment consisting of a special diet low in methionine, aspirin, and vitamin B6 or cystine can prevent mortality and reduce other clinical features.
- CF** **Cystic Fibrosis;** defect in the cystic fibrosis transmembrane conductor regulator (CFTR) gene which regulates the body's salt and water secretions. This results in the build up of thick, sticky mucus in the lungs and digestive system. Treatment consists of pancreatic enzymes, vitamin supplements, chest physiotherapy, and antibiotics. Early treatment improves physical growth and cognitive function, and possibly lung function.

**Table I: Births by County of Occurrence –
Infants Detected by County of Residence**

COUNTY	2007 BIRTHS	2007 INFANTS DETECTED										ALL INFANTS
		PKU	CH	CAH	Hb	GAL	BIO	MCAD	MSUD	HCY	CF	
Adams	594	0	0	0	0	0	0	0	0	0	0	0
Asotin	1	0	0	0	0	0	0	0	0	0	0	0
Benton	4,057	0	1	0	1	0	0	0	0	0	0	2
Chelan	1,552	0	1	0	0	0	0	0	0	0	0	1
Clallam	612	0	0	0	0	0	0	0	0	0	0	0
Clark	5,818	0	1	0	0	0	0	1	0	0	0	2
Columbia	0	0	0	0	0	0	0	0	0	0	0	0
Cowlitz	1,315	0	0	0	0	0	0	0	0	0	0	0
Douglas	0	0	0	0	0	0	0	0	0	0	0	0
Ferry	5	0	0	0	0	0	0	0	0	0	0	0
Franklin	372	0	1	0	0	0	0	0	0	0	0	1
Garfield	0	0	0	0	0	0	0	0	0	0	0	0
Grant	1,196	0	0	0	0	0	0	0	0	0	0	0
Grays Harbor	585	0	0	0	0	0	0	0	0	0	1	1
Island ^a	263	0	0	0	0	0	0	0	0	0	0	0
Jefferson	130	0	0	0	0	0	0	0	0	0	0	0
King	29,810	3	29	2	18	3	0	2	0	0	3	60
Kitsap ^a	2,080	0	1	0	0	0	0	0	0	0	1	2
Kittitas	333	0	0	0	0	0	0	0	0	0	0	0
Klickitat	98	0	0	0	0	0	0	0	0	0	0	0
Lewis	666	0	0	0	0	1	0	0	0	0	0	1
Lincoln	0	0	0	0	0	0	0	0	0	0	0	0
Mason	294	0	0	0	0	0	0	0	0	0	0	0
Okanogan	521	0	0	0	0	0	0	0	0	0	0	0
Pacific	7	0	0	0	0	0	0	0	0	0	0	0
Pend Oreille	113	1	0	0	0	0	0	0	0	0	0	1
Pierce ^a	9,432	0	5	1	3	1	0	0	0	0	5	15
San Juan	3	0	0	0	0	0	0	0	0	0	0	0
Skagit	1,723	0	1	0	0	1	0	0	0	0	0	2
Skamania	1	0	0	0	0	0	0	0	0	0	0	0
Snohomish	6,157	1	1	0	1	1	1	1	0	0	0	6
Spokane	6,932	0	3	2	0	0	0	1	0	0	1	7
Stevens	286	0	0	0	0	0	0	0	0	0	0	0
Thurston	3,087	0	0	0	0	0	0	0	0	0	1	1
Wahkiakum	0	0	0	0	0	0	0	0	0	0	0	0
Walla Walla	859	0	0	0	0	0	0	0	0	0	0	0
Whatcom	2,187	0	1	0	0	0	0	0	0	0	0	1
Whitman	431	0	0	0	0	0	0	0	0	0	0	0
Yakima	4,347	2	3	0	0	0	0	1	0	0	0	6
TOTAL^a	85,867	7	48	5	23	7	1	6	0	0	12	109

^a Excludes infants born in military hospitals that do not participate in the Newborn Screening Program (2,033 born at Madigan Army Medical Center, 450 born at Oak Harbor Naval Hospital and 624 born at Bremerton Naval Hospital). Total excluded = 3,077.

Table II: Births and Infants Detected by Infant's Race

INFANTS RACE ^a	2007 BIRTHS	2007 INFANTS DETECTED ^a										ALL INFANTS
		PKU	CH	CAH	Hb	GAL	BIO	MCAD	MSUD	HCY	CF	
White	66,795	7	21	5	1	7	1	5	0	0	12	59
African American	5,811	0	2	0	8	0	0	0	0	0	0	10
Asian	9,629	0	20	0	8	0	0	0	0	0	0	28
Native American	2,689	0	0	0	0	0	0	0	0	0	0	0
Unknown/Other ^c	943	0	5	0	6	0	0	1	0	0	0	12
TOTAL^b	85,867	7	48	5	23	7	1	6	0	0	12	109

Hispanic ^d	19,934	0	6	1	2	1	1	3	0	0	0	14
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^a The infant's race for 2007 is from birth certificate data and was determined by an algorithm of mother and father's race developed by the National Center for Health Statistics. The race of infants detected is from information provided on the newborn screening test form.

^b Excludes infants born in military hospitals that do not participate in the Newborn Screening Program (2,033 born at Madigan Army Medical Center, 450 born at Oak Harbor Naval Hospital and 624 born at Bremerton Naval Hospital). Total excluded = 3,077.

^c Includes multiracial (more than one race designation on the screening form) or unknown (no designation made).

^d Hispanics can be of any race; they are included in the figures above.

Newborn Screening Follow-Up Procedures

All specimens that are determined to be presumptive positive through the Newborn Screening Program are followed up immediately through direct telephone contact with the child's physician. This is to ensure that diagnostic testing and treatment, if indicated, is initiated as quickly as possible. Following a definitive diagnosis, a long-term, disease-specific medical management program is implemented as follows:

Phenylketonuria (PKU) - Children are seen monthly in Seattle and every other month in Spokane by the Department of Health (DOH) subsidized University of Washington Phenylketonuria Clinic. An interdisciplinary team consisting of a pediatric metabolic physician, nutritionists, social worker, and genetic counselor work closely with each family to optimize the child's dietary compliance through intensive education and support services. Periodic neuropsychological assessments are also provided for all patients to monitor cognitive and emotional development. For adults with PKU, consultative, support and nutritionist management services are provided at the University of Washington Division of Metabolism and Nutrition. Critical reproductive counseling and maternity services for women at risk of fetal damage due to Maternal PKU are also available. Other program components include a summer camp and Science Night through the private, non-profit PKU Action Group.

Congenital Hypothyroidism (CH) - Thyroid hormone therapy is monitored by the child's primary health care provider and/or pediatric endocrinologist. The DOH subsidized Congenital Hypothyroidism Developmental Evaluation Clinic at the University of Washington provides developmental, neuropsychological and occupational therapy assessments for affected children.

Congenital Adrenal Hyperplasia (CAH) - All children are seen for a diagnostic work-up by a pediatric endocrinologist to establish appropriate steroid hormone therapy. Long-term management is monitored by the child's primary health care provider and/or pediatric endocrinologist. Affected females with genital abnormalities related to the disorder are referred for surgical consultation.

Sickle Cell Disease (SCD) - Affected children are administered prophylactic penicillin and folic acid. Their long-term management is provided by a pediatric hematologist or an interdisciplinary team consisting of a pediatric hematologist, nurse, social worker and genetic counselor at a DOH subsidized Comprehensive Sickle Cell Clinic – Children's Hospital Odessa Brown Center (Seattle) or Mary Bridge Children's Center (Tacoma). The clinic staff work closely with each family to optimize the child's health and well-being through intensive education and support services. Periodic neuropsychological assessments are also provided for all patients to monitor cognitive and emotional development. Other sickle cell disease program components include a summer camp and other educational and support activities through the DOH supported Northwest Sickle Cell Collaborative.

Galactosemia, Biotinidase deficiency, MCAD deficiency, Maple Syrup Urine Disease, and Homocystinuria – All children with these disorders are seen periodically as needed by the Department of Health (DOH) subsidized University of Washington Biochemical Genetics Clinic at the University of Washington in Seattle, with a quarterly satellite in Spokane. Like PKU, an interdisciplinary team consisting of a pediatric metabolic physician, nutritionists, social worker, and genetic counselor work closely with each family to optimize the child's dietary compliance through intensive education and support services. Periodic neuropsychological assessments are also provided for all patients to monitor cognitive and emotional development.

Cystic Fibrosis (CF) – All children with cystic fibrosis are seen periodically as needed by one of the four regional CF Foundation accredited Clinics – Children's Hospital (Seattle), Mary Bridge (Tacoma), Deaconess Hospital (Spokane), or Oregon Health Sciences University (Portland). Like PKU, an interdisciplinary team consisting of a pediatric pulmonologist, nutritionist, social worker, and nurse work closely with each family to optimize the child's growth and minimize medical complications of the condition, particularly lung disease.

Table III: Follow-Up Status of Infants Detected (Severe Disease)

FOLLOW-UP	2007 INFANTS DETECTED										ALL INFANTS
	PKU	CH	CAH	SCD	GAL	BIO	MCAD	MSUD	HCY	CF	
Followed by medical specialist – (i.e., pediatric endocrinologist, hematologist, or comprehensive clinic)	5	30	3	8	1	0	4	0	0	12	63
Followed by primary care provider, with some consultation from specialist	0	2	0	0	0	0	0	0	0	0	2
Expired or Lost to Follow-up	0	0	1 ^a	1 ^b	0	0	0	0	0	0	2
TOTAL	5	32	4	9	1	0	4	0	0	12	67

^a Military baby moved to Kansas, diagnosis confirmed but status of clinical management unknown.

^b Baby died at 14 days of age (cause unrelated to sickle cell disease).

Table IV: Age at which Treatment Began for Infants Detected (Severe Disease)

DISORDER	NUMBER OF INFANTS	AGE TREATMENT BEGAN (DAYS)	
		AVERAGE	RANGE
PKU	5	7	5 – 8
CH	32	16	4 – 50 ^a
CAH	4	15	3 – 29 ^b
SCD	8 ^c	30	18 – 49
GAL	1	4	n/a
BIO	0	n/a	n/a
MCAD	4	9	6 – 12
MSUD	0	n/a	n/a
HCY	0	n/a	n/a
CF	12	24	1 - 52

^a Includes ten infants who weren't detected until their second screen.

^b Includes two infants who weren't detected until their second screen.

^c Excludes baby who died (as noted above).

Screening Costs 2007

The department's cost to operate the program, including laboratory testing, monitoring to assure adequate screening for all infants, follow up of all abnormal findings, education, and evaluation, is covered through a fee charged for each infant through the facility of birth. For the period covered, the charge was \$60.90 for each child.

In addition to the screening fee, a separate charge of \$3.50 per birth was collected during this period to support specialty clinic care for infants diagnosed through newborn screening.

An additional \$3.10 fee to support specialty clinic care was authorized by the 2005 legislature and implemented in November 2005. The \$3.10 fee expired on June 30, 2007. Lost revenue from this fee was replaced with state general funds by the 2007 legislature.

Preliminary Data for 2008 – Infants Detected

In 2008, approximately 86,320 infants were screened by the Washington State Newborn Screening Program. This excludes approximately 3,000 infants born at the three Washington military hospitals.

DISORDER	INFANTS DETECTED
Amino Acid Disorders	9
Biotinidase Deficiency	0
Congenital Adrenal Hyperplasia (CAH)	8
Congenital Hypothyroidism (CH)	83
Cystic Fibrosis	16
Fatty Acid Oxidation Disorders	5
Galactosemia	2
Hemoglobinopathies ^a	15
Organic Acid Disorders	0
TOTAL	138

^a Over 1,000 additional benign hemoglobin conditions and traits were detected; trait requires no treatment but may be informative for family planning.

APPENDIX A: Newborn Hemoglobin Screening – Explanation and Definitions of Phenotypes Found

Hemoglobins are by far the most complex of the conditions detected by newborn screening. More than a dozen genes are involved in hemoglobin production and nearly 700 abnormalities have been described by researchers and clinicians. Also, a variety of combinations are possible for any individual. Many of the abnormalities are rare and most have no clinical implications. A primary objective of our program is to identify those infants with sickle cell disease because these infants will suffer far less illness and death if they are promptly entered into a comprehensive health care program that includes prophylactic treatment with penicillin.

PHENOTYPE	MOST LIKELY GENOTYPE/CLINICAL IMPLICATIONS
FSS	Homozygous for hemoglobin S. Results in sickle cell anemia, a severe form of sickle cell disease.
FSC	Hemoglobin S in combination with hemoglobin C. Results in sickle C disease, a moderate to severe form of sickle cell disease.
FSA	Hemoglobin S in combination with β -thalassemia ^a minor. A moderate form of sickle cell disease.
FSE	Hemoglobin S in combination with hemoglobin E. Results in sickle E disease, a mild to moderate form of sickle cell disease.
FVS	Hemoglobin S in combination with an unidentified hemoglobin variant. A mild to moderate form of sickle cell disease depending upon the particular variant.
FE–	Hemoglobin E in combination with β -thalassemia ^a major. A moderate to severe hemolytic anemia.
FE– + Bart's	Hemoglobin E in combination with β -thalassemia ^a major and α -thalassemia ^b genes. A moderate to severe hemolytic anemia.
FA+CS+ High Bart's	Two hemoglobins (Constant Spring and Bart's) indicative of multiple α -thalassemia ^b genes. A moderate to severe anemia.
FAA + High Bart's	High level of hemoglobin Bart's indicative of multiple α -thalassemia ^b genes. Likelihood of Hemoglobin H disease, a moderate to severe hemolytic anemia.
FAE + High Bart's	Hemoglobin E trait in combination with a high level of hemoglobin Bart's indicative of multiple α -thalassemia ^b genes. Likelihood of Hemoglobin H disease, a moderate to severe hemolytic anemia.
FVA	An unidentified hemoglobin variant with β -thalassemia ^a minor. A mild to moderate anemia depending upon the particular variant.

^a Decreased production of β globin chains; benign to severe anemia. Significant interaction with other β chain variants such as hemoglobin S, E, and D.

^b Decreased production of α globin chains; benign to severe anemia depending on how many of the four α globin genes are affected.

PHENOTYPE	MOST LIKELY GENOTYPE/CLINICAL IMPLICATIONS
FCC	Homozygous for hemoglobin C. A mild to moderate hemolytic anemia.
FCA	Hemoglobin C in combination with β -thalassemia ^a minor. A mild to moderate hemolytic anemia.
FDA	Hemoglobin D in combination with β -thalassemia ^a minor. A mild to moderate hemolytic anemia.
FDD	Homozygous for hemoglobin D. A mild to moderate hemolytic anemia.
FEE	Homozygous for hemoglobin E. Mild anemia.
FEE+Bart's	Homozygous hemoglobin E in combination with α -thalassemia ^b . Mild anemia.
FAE+CS+Bart's	Hemoglobin E trait in combination with two hemoglobins (Constant Spring and Bart's) indicative of α -thalassemia ^b genes. Mild anemia.
FA+CS+Bart's	Two hemoglobins (Constant Spring and Bart's) indicative of α -thalassemia ^b genes. Mild anemia.
FAS+Bart's	Hemoglobin S trait in combination with α -thalassemia ^b . No clinical implications for S trait (see FAS, below). Benign to mild anemia.
FAE+Bart's	Hemoglobin E trait in combination with α -thalassemia ^b . No clinical implications for E trait (see FAE, below). Benign to mild anemia.
FAC+Bart's	Hemoglobin C trait in combination with α -thalassemia ^b . No clinical implications for C trait (see FAC, below). Benign to mild anemia.
FAA+Bart's	α -thalassemia ^b . Benign to mild anemia.
FAS	Hemoglobin S trait. No clinical implications for child. Family may be at risk for sickle cell disease.
FAE	Hemoglobin E trait. No clinical implications for child. Family may be at risk for homozygous E or hemoglobin E/ β -thalassemia ^a , a significant hemoglobin disease.
FAC	Hemoglobin C trait. No clinical implications for child. Family may be at risk for homozygous C, a mild to moderate hemolytic anemia or hemoglobin SC, a moderate to severe form of sickle cell disease.
FAD	Hemoglobin D trait. No clinical implications for child. Homozygous state is benign; however, family may be at risk for hemoglobin SD, a moderate to severe form of sickle cell disease.
FA+Var	Unidentified variant trait. Clinical effects unlikely.

^a Decreased production of β globin chains; benign to severe anemia. Significant interaction with other β chain variants such as hemoglobin S, E, and D.

^b Decreased production of α globin chains; benign to severe anemia depending on how many of the four α globin genes are affected.

APPENDIX B: Newborn Hemoglobin Screening – Infants Detected by Phenotype and Race/Ethnicity

January through December 2007; Number of Infants = 85,867

PHENOTYPE	TOTAL	WHITE	BLACK	ASIAN	NAT. AMER.	OTHER ^a	HISPANIC ^b
FSS	4	0	4	0	0	0	0
FSC	5	0	2	0	0	3	1
FSA	2	0	2	0	0	0	0
FSE	1	0	0	0	0	1	0
FVS	1	0	0	0	0	1	0
FE-	4	0	0	4	0	0	1
FE- + Bart's	1	0	0	1	0	0	0
FAE+CS+High Bart's	1	0	0	1	0	0	0
FAA+High Bart's	4	0	0	4	0	0	0
FAE+High Bart's	1	1	0	0	0	0	0
FVA	1	0	0	0	0	1	0
FCC & FCA	2	0	2	0	0	0	0
FDA & FDD	3	1	0	0	0	2	0
FEE	11	0	0	11	0	0	0
FEE+Bart's	1	0	0	1	0	0	0
FAE+CS+Bart's	9	2	1	4	0	2	0
FA+CS+Bart's	14	0	0	13	0	1	2
FAS+Bart's	10	1	6	1	1	1	0
FAE+Bart's	20	4	0	15	0	1	0
FAC+Bart's	3	1	1	1	0	0	0
FAA+Bart's	196	41	36	107	1	11	10
FAS	529	178	294	22	15	20	80
FAE	289	51	9	205	3	21	11
FAC	90	28	56	3	1	2	10
FAD	37	22	4	4	2	5	5
FA+Var	164	111	9	21	5	18	27
TOTAL	1403	441	426	418	28	90	147

^a Includes multi-racial (more than one race designation on the screening form) or unknown (no designation made).

^b Hispanics can be of any race; they are included in figures to the left.