

## DEPARTMENT OF HEALTH

NOTICE OF FINAL RULEMAKING

The Director of the Department of Health, pursuant to the authority set forth in § 5 of the Newborn Hearing Screening Act of 2000, effective April 4, 2001 (D.C. Law 13-276; D.C. Official Code § 7-854), Mayor's Order 2002-12, dated January 25, 2002, § 4 of the District of Columbia Newborn Screening Requirement Act of 1979, effective April 29, 1980 (D.C. Law 3-65; D.C. Official Code § 7-833 ), and Mayor's Order 2004-172, dated October 20, 2004, hereby gives notice of the adoption of the following amendments to Title 22 of the District of Columbia Municipal Register (DCMR). The Director took final action to adopt the rules on September 14, 2009. A Notice of Emergency and Proposed Rulemaking was published on August 7, 2009, at 56 DCR 6229. No comments were received in response to the notice, and no changes have been made to the rules since publication of the Notice of Emergency and Proposed Rulemaking.

The final rules reinstate neonatal screening services rules formerly codified in sections 2099 and 2204 of Title 22 by establishing a new Chapter 21, and make corresponding technical amendments in section 2600.8, which cross-references the neonatal screening provisions. The rules will become effective upon publication of this notice in the *D.C. Register*.

**Title 22 (Public Health and Medicine) (August 1986) of the DCMR is amended as follows:**

**Add a new Chapter 21 to read as follows:**

**CHAPTER 21 NEONATAL SCREENING SERVICES****2100 NEWBORN HEARING SCREENING**

- 2100.1 Each institution shall establish a Newborn Hearing Screening Service according to the following requirements:
- (a) Each institution shall designate a person to be responsible for the newborn hearing screening service in that institution.
  - (b) An audiologist, otolaryngologist, or other qualified person, including a neonatal nurse or a hospital technician, shall oversee each newborn hearing screening service. The person assigned to oversee the newborn hearing screening service may be full or part time, on or off site, an employee of the hospital, or under contract or other arrangement that allows him or her to oversee the newborn hearing screening service. This person shall advise the institution about all aspects of the newborn hearing screening service,

including screening, and recommendations for follow-up testing and treatment.

- (c) Each institution shall provide hearing screening services pursuant to this section, unless any of the following occurs:
  - (1) The procedure is contrary to the parents' religious beliefs;
  - (2) The parents withhold consent to perform the screening; or
  - (3) The institution transfers the newborn to another institution for treatment before hearing screening can be completed, provided that the transferring institution informs the Perinatal and Infant Health Bureau of the Department, or its successor, within twenty-four (24) hours.
- (d) Newborn hearing screening may be performed by any of the following:
  - (1) An audiologist;
  - (2) An otolaryngologist;
  - (3) A neonatal nurse appropriately trained to perform hearing screening and under supervision by an audiologist or otolaryngologist;
  - (4) A hospital technician appropriately trained to perform hearing screening and under supervision by an audiologist or otolaryngologist; or
  - (5) A hospital volunteer appropriately trained to perform hearing screening and under supervision by an audiologist or otolaryngologist.

2100.2 Before discharging the newborn, each institution shall do the following:

- (a) Provide the newborn's parents with oral information and written materials that describe the benefits and purpose of hearing screening, the procedures used for hearing screening, and the consequences of hearing loss;
- (b) Provide the newborn's parents with oral and written information about whether it performed a hearing screening on the newborn;

- (c) After performing the hearing screening, provide the newborn's parents, the newborn's primary care provider, if known, and the Perinatal and Infant Health Bureau of the Department, or its successor, with oral and written results of the hearing screening; and
- (d) After performing the hearing screening, recommend to the newborn's parents and the newborn's primary care provider, if known, appropriate follow-up testing and treatment that may be necessary.

2100.3 If the parents do not understand English well enough to comprehend the information, the institution shall provide the information required by § 2100.2 in the parents' native language.

2100.4 For newborns that require additional procedures to complete the screening after being discharged from the institution, the institution shall provide the newborn's parents and the newborn's primary care provider, if known, with written notice about the availability and importance of additional screening procedures.

2100.5 An institution that completes a newborn hearing screening and finds that the newborn did not pass the screening shall provide the newborn's parents, the Department, and the newborn's primary care provider, if known, with written results of the screening, recommended diagnostic procedures, and resources available for newborns with hearing impairment.

## **2101 METABOLIC DISORDERS**

**2101.1** Each institution shall make available blood tests to screen each newborn delivered or cared for at the institution for the following metabolic disorders:

- (a) 2,4-Dienoyl-CoA reductase deficiency;
- (b) 2-Methylbutyl-CoA dehydrogenase deficiency;
- (c) 3-Methylcrotonyl-CoA carboxylase deficiency (3MCC);
- (d) 3-Methylglutaconyl-Coa hydratase deficiency;
- (e) 3-OH 3-CH3 glutaric aciduria (HMG);
- (f) 5-Oxoprolinuria (pyroglutamic aciduria);
- (g) Argininemia;

- (h) Argininosuccinic acidemia (ASA);
- (i) Beta-ketothiolase deficiency (BKT);
- (j) Biotinidase deficiency (BIOT);
- (k) Carbamoylphosphate synthetase deficiency (CPS def.);
- (l) Carnitine uptake defect (CUD);
- (m) Citrullinemia (CITR);
- (n) Congenital adrenal hyperplasia (CAH);
- (o) Cystic fibrosis (CF);
- (p) Galactosemia (GALT);
- (q) Glucose-6-Phosphate Dehydrogenase Deficiency (G6PD);
- (r) Glutaric acidemia type I (GA I);
- (s) Hemoglobinopathy;
- (t) Homocystinuria (HCY);
- (u) Hyperammonemia, hyperornithinemia, homocitrullinemia syndrome (HHH)
- (v) Hyperornithine with gyral atrophy;
- (w) Hypothyroidism;
- (x) Isobutyryl-CoA dehydrogenase deficiency;
- (y) Isovaleric acidemia (IVA);
- (z) Long-chain L-3-OH acyl-CoA dehydrogenase deficiency (LCHADD);
- (aa) Malonic aciduria;
- (bb) Maple Syrup Urine Disease (MSUD);
- (cc) Medium chain acyl-CoA dehydrogenase deficiency (MCAD);

- (dd) Methylmalonic acidemia (Cbl A, B);
  - (ee) Multiple acyl-CoA dehydrogenase deficiency (MADD);
  - (ff) Multiple carboxylase deficiency (MCD);
  - (gg) Neonatal carnitine palmitoyl transferase deficiency-type II (CPT-II);
  - (hh) Phenylketonuria (PKU);
  - (ii) Propionic acidemia (PROP);
  - (jj) Short chain acyl-CoA dehydrogenase deficiency (SCAD);
  - (kk) Short chain hydroxy acyl-CoA dehydrogenase deficiency (SCHAD);
  - (ll) Trifunctional protein deficiency (TFP);
  - (mm) Tyrosinemia type I (TYR I); and
  - (nn) Very long-chain acyl-CoA dehydrogenase deficiency (VLCAD).
- 2101.2 Each institution shall inform the parent or parents of the availability and purpose of the tests for the conditions set forth in subsection 2201.1 and shall document in the newborn's health record that the parent or parents were properly informed and understood the purpose of the tests.
- 2101.3 Each institution shall provide the parent or parents a reasonable opportunity to object to performance of the tests and shall document in the newborn's health record whether the parent or parents consented or withheld consent to have the testing done.
- 2101.4 Each institution that has obtained parental consent to have the newborn tested shall take from the newborn a blood sample of sufficient quantity to enable a laboratory designated by the Mayor to analyze the sample for the tests identified in subsection 2201.1, unless an identical test has been performed. Each institution shall send the sample to the designated laboratory.
- 2101.5 A newborn's parent is indigent for the purpose of § 9 of the District of Columbia Newborn Screening Requirement Act of 1979, effective April 29, 1980 (D.C. Law 3-65; D.C. Official Code § 7-838), if the parent does not have coverage by Medicaid or third party medical or health insurance coverage and has total pre-tax household income, including child support payments, alimony, rent payments received, and any other income

received on a regular basis, equal to or less than three hundred per cent (300%) of the federal poverty level.

2101.6 A newborn's parent shall document income to satisfy the requirements of § 2101.5 as follows:

- (a) For a person whose source of income is earned income, one of the following:
  - (1) Originals or copies of all earnings statements received within the previous thirty (30) days;
  - (2) A copy of the first two (2) pages of a District of Columbia tax return for the most recent tax year;
  - (3) A copy of the first page of a Federal tax return for the most recent tax year; or
  - (4) For a newly employed parent, a copy of an offer of employment that states the amount of salary to be paid.
- (b) For a parent whose source of income includes unearned income, one of the following:
  - (1) A copy of a Social Security or worker's compensation benefit statement;
  - (2) Proof of child support or alimony received; or
  - (3) A copy of a Federal tax return for the most recent tax year, including all schedules and attachments.

2101.7 A newborn is a resident of the District of Columbia for the purpose of § 9 of the District of Columbia Newborn Screening Requirement Act of 1979, effective April 29, 1980 (D.C. Law 3-65; D.C. Official Code § 7-838), if the newborn's mother is a resident of the District of Columbia on the date on which the newborn was born.

2101.8 The mother of a newborn shall document residency to satisfy the requirements of § 2101.7 by providing one of the following:

- (a) A valid motor vehicle operator's permit issued by the District;
- (b) A non-driver's identification card issued by the District;

- (c) A voter registration card issued by the District of Columbia Board of Elections and Ethics:
- (d) A copy of a lease or a rent receipt for real property located in the District;
- (e) A utility bill for real property located in the District; or
- (f) A copy of the most recent Federal income tax return or Earned Income Credit Form.

## 2199 DEFINITIONS

2199.1 When used in this chapter, the following terms and phrases shall have the meanings ascribed:

**2,4-Dienoyl-CoA reductase deficiency**—an autosomal recessive genetic disorder characterized by a deficiency of 2,4 Dienoyl CoA Reductase necessary for the degradation of unsaturated fatty acids with an even number of double bonds. Symptoms include sepsis, hypotonia, decreased feeding, and intermittent vomiting. Low carnitine levels can be detected and respiratory acidosis may occur.

**2-Methylbutryl-CoA dehydrogenase deficiency**—an autosomal recessive genetic disorder resulting from a defect in the metabolism of the branched chain amino acid isoleucine. Symptoms include poor feeding, lethargy, hypoglycemia, and metabolic acidosis. Symptomatic patients display developmental delay, seizure disorders, and progressive muscle weakness in infancy and childhood.

**3-Methylcrotonyl-CoA carboxylase deficiency (3MCC)**—a progressive autosomal recessive genetic disorder characterized by failure to thrive, hypotonia, muscle atrophy, seizures, mental retardation, and dermatological changes.

**3-Methylglutaconyl-CoA hydratase deficiency**—an autosomal recessive genetic disorder involving an enzyme in the metabolism of the amino acid leucine. Symptoms appear in a wide range of clinical severity and may include acute life-threatening cardiopulmonary symptoms soon after birth, psychomotor retardation, hypotonia, failure to thrive, microcephaly, seizures, and spasticity. Some patients may have acute episodes of vomiting, metabolic acidosis, and lethargy progressing to coma.

**3-OH 3-CH<sub>3</sub> glutaric aciduria or 3-hydroxy-3-methylglutaryl-CoA lyase deficiency (HMG)**—an autosomal recessive disorder. Symptoms may include metabolic acidosis, hypoglycemia, sensitivity to dietary leucine, carnitine deficiency, hepatomegaly, fever, somnolence, and coma. If this disorder is untreated, it is likely to result in death during childhood.

**5-Oxoprolinuria (pyroglutamic aciduria)**—a group of autosomal recessive genetic conditions including glutathione synthetase deficiency, glutamylcysteine synthetase deficiency and 5-oxoprolinase deficiency caused by a deficiency of one (1) of three (3) enzymes in the gamma glutamyl cycle and characterized by metabolic acidosis, hemolytic anemia, electrolyte imbalance, and jaundice.

**Argininemia**—an autosomal recessive genetic condition that presents from two (2) months to four (4) years of age. Symptoms include progressive spastic paraplegia, failure to thrive, delayed milestones, hyperactivity, and irritability, with episodic vomiting, hyperammonemia, seizures, microcephaly, and cerebral atrophy resulting in mental retardation.

**Argininosuccinic acidemia (ASA)**— an autosomal recessive disorder of the urea cycle. Symptoms are hyperammonemia accompanied by lack of appetite, vomiting, listlessness, seizures, and coma. Onset is usually at birth, but symptoms may not be noticeable for days or weeks. The build up in ASA, if too high, ultimately causes a build up in ammonia. Build up of ammonia is toxic and can cause brain damage. ASA is also characterized by excessive urinary excretion of argininosuccinic acid, epilepsy, ataxia, mental retardation, liver disease, and friable, tufted hair.

**Audiologist**—a person who meets the education and experience requirements for a Certificate of Clinical Competence in the area of audiology granted by the American Speech and Hearing Association or who meets the educational requirements for certification and is in the process of accumulating the supervised experience required for certification.

**Beta-ketothiolase deficiency (BKT)**—an autosomal recessive disorder characterized by recurrent severe metabolic acidosis. Symptoms include increased plasma glycine level, metabolic acidosis, episodic ketosis, vomiting, dehydration, coma, and cardiomyopathy, with on average onset of five (5) to twenty-four (24) months.

**Biotinidase deficiency (BIOT)**—an autosomal recessive disorder characterized by a lack of the enzyme biotinidase that can lead to seizures, developmental delay, eczema, and hearing loss that are treated with free biotin. Symptoms include hypotonia, ataxia, alopecia, seborrheic dermatitis, and optic nerve atrophy. Metabolic acidosis can result in coma and death.

**Carbamoylphosphate synthetase deficiency (CPS def.)**—an autosomal recessive genetic condition that presents within seventy-two (72) hours with symptoms of lethargy, vomiting, hypothermia, respiratory alkalosis, and seizures progressing to coma. Survivors of the newborn period have recurrent episodes of hyperammonemia associated with viral infections or increased dietary protein intake. Some patients have a later onset with less severe symptoms.

**Carnitine uptake defect (CUD)**—a class of autosomal recessive disorders characterized by hypoketotic hypoglycemia, seizures, vomiting, lethargy progressing to coma,

cardiomyopathy, and hepatomegaly. This disorder includes carnitine palmitoyl transferase deficiency type I and carnitine acylcarnitine translocase deficiency.

**Citrullinemia (CITR)**—an autosomal recessive genetic disorder characterized by a deficiency of argininosuccinic acid synthetase, hyperammonemia accompanied by lack of appetite, vomiting, listlessness, seizures, and coma. Onset is usually at birth, but symptoms may not be noticeable for days or weeks. When left untreated, brain damage, coma, and death will occur.

**Congenital adrenal hyperplasia (CAH)**— a set of inherited disorders that occurs in both males and females as a result of the excess production of male hormones and an underproduction of the enzyme 21-hydroxylase. Symptoms include severe acne, excess facial or body hair, early development of pubic hair, receding scalp hairline, menstrual disturbances in females, and infertility in males and females in its mild form and ambiguous genitalia in newborn girls and salt and hormonal imbalances in girls and boys in more severe forms. If not treated, CAH can cause heart failure and death within a few days from birth. CAH can not be cured, however, it can be effectively treated.

**Cystic fibrosis (CF)**—an autosomal recessive disorder characterized by progressive chronic damage to the respiratory system, chronic digestive system problems, and can affect other organs. CF affects mucus-producing glands producing thick mucus that can obstruct air passages in the lungs, affects sweat and salivary glands, and blocks enzymes secreted by the pancreatic duct. Cystic Fibrosis can cause lung disease, failure to grow, clubbed fingers and toes, muscular weakness, and visual impairment.

**Department**—the District of Columbia Department of Health.

**Director**—the Director of the District of Columbia Department of Health.

**Galactosemia**—a condition involving the inability to convert galactose to glucose.

**Glucose-6-phosphate dehydrogenase deficiency (G6PD)**—a condition resulting in anemia or jaundice that is made worse by certain medications and some foods.

**Glutaric acidemia type I (GA-I)**—an autosomal recessive enzyme deficiency genetic disorder characterized by hypoglycemia, dystonia, and dyskinesia. After a period of apparently normal development, the disorder may appear suddenly and present as vomiting, metabolic acidosis, hypotonia, and central nervous system degeneration. It is not yet known how or why Glutaric Acid causes brain damage, yet damage occurs when a crisis causes an acidic environment in the blood created by excess protein byproducts. Crises can be provoked by common childhood illnesses such as colds, flu, ear infections, stomach virus, fever, etc.

**Hearing impairment**—a dysfunction of the auditory system, of any type or degree, sufficient to interfere with the acquisition and development of speech and language skills, with or without the use of sound amplification.

**Hearing screening**—an objective physiological measure of hearing sensitivity used to determine the likelihood of hearing loss.

**Hemoglobinopathy**—a class of disorders caused by the presence of abnormal hemoglobin production in the blood, due to genetic variations that can result in production of hemoglobin with different structures or thalassemias and reduction in the amount of normal hemoglobin produced. This term includes the following hemoglobin variants: HbS, HbC, HbE, HbD, and alpha/beta thalassemias.

**Homocystinuria**—a condition resulting from one of several genetically determined errors of methionine metabolism.

**Hyperammonemia, hyperornithinemia, homocitrullinemia syndrome (HHH)**—an autosomal recessive genetic disorder that may present at birth or in later childhood. Newborns on high protein formulas or foods may vomit with feeding, refuse to eat, become lethargic, or develop hyperammonemic coma. Patients gravitate to diets low in milk and meat during childhood.

**Hyperornithine with gyrate deficiency**—an autosomal recessive genetic disorder characterized by slow progressive vision loss leading to blindness. Myopia and decreased night vision appear as early symptoms in the patient's teens and early twenties.

**Hypothyroidism**—those clinical conditions that result from abnormally low circulating levels of thyroid hormone.

**Institution**—a hospital or maternity center.

**Isobutyryl-CoA dehydrogenase deficiency**—an autosomal recessive genetic disorder involving the inability to metabolize valine with a highly variable presentation.

**Isovaleric acidemia (IVA)**—an autosomal recessive genetic disorder caused by a defect in the breakdown of the molecule isovaleryl-CoA that presents in acute or intermittent episodes. IVA can present as an acute episode of illness during the first few weeks of a newborn's life, or it may present chronically with intermittent episodes of illness throughout life. Both forms of IVA are caused by the same biochemical defect. Infants who survive an acute neonatal episode will go on to exhibit the chronic intermittent form. Symptoms of acute IVA are attacks of vomiting, lack of appetite, and listlessness; lethargy, neuromuscular irritability, and hypothermia are other characteristics. Episodes can be triggered by upper respiratory infections or by excessive consumption of high-protein foods. Early detection through newborn screening and good treatment of IVA generally leads to normal development. Permanent neurologic damage can occur if an acute episode is not prevented or is misdiagnosed.

**Long-chain L-3-OH acyl-CoA dehydrogenase deficiency (LCHADD)**—an autosomal recessive genetic disorder characterized by failure to oxidize fatty acids due to a missing

or malfunctioning enzyme. Symptoms include hypoglycemia, lethargy, failure to thrive, cardiomyopathy and developmental delay. Early identification and treatment can prevent life-threatening episodes.

**Malonic aciduria**—an autosomal recessive genetic disorder caused by a deficiency of malonyl-CoA decarboxylase (MCD) with a variable presentation ranging from acute neonatal onset to later in childhood. Symptoms include developmental delay, seizures, hypotonia, diarrhea, vomiting, metabolic acidosis, hypoglycemia, and ketosis.

**Maple syrup urine disease (MSUD)**—a condition resulting from the impairment of branched chain alpha-ketoacid dehydrogenase.

**Maternity center**—a facility or other place, other than a hospital or the mother's home, that provides antepartal, intrapartal, and postpartal care for both mother and newborn infant during and after normal, uncomplicated pregnancy.

**Medium chain acyl-CoA dehydrogenase deficiency (MCADD)**—an autosomal recessive genetic disorder characterized by inability to convert fat to energy. Fasting is not tolerated well in people with MCADD. Symptoms generally begin in infancy or early childhood, however, there are some with no apparent symptoms at birth. Low blood sugar, seizures, brain damage, cardiac arrest and serious illness can occur very quickly in children who are not feeding well. Some experience recurrent episodes of metabolic acidosis, hypoglycemia, lethargy, and coma. If not detected and treated appropriately, MCADD can result in intellectual and developmental disability and death. Those treated are expected to have normal life expectancy.

**Metabolic disorder**—a disorder that results in a defect in the function of a specific enzyme or protein.

**Methylmalonic acidemia**—one (1) of two (2) variations of an autosomal recessive genetic disorder caused by an enzymatic defect in the oxidation of amino acids characterized by lethargy, failure to thrive, vomiting, dehydration, respiratory distress, hypotonia, and hepatomegaly. Acute episodes may include drowsiness, coma, and seizures, with subsequent developmental delays. This disorder includes methylmalonic acidemia CblA, methylmalonic acidemia CblB, and methylmalonic acidemia mutase deficiency.

**Multiple acyl-CoA dehydrogenase deficiency (MADD)**—an autosomal recessive genetic disorder, also known as glutaric acidemia type II, with three (3) different clinical presentations. Symptoms include hypotonia, hepatomegaly, severe nonketotic hypoglycemia, metabolic acidosis, and variable body odor of sweaty feet.

**Multiple carboxylase deficiency (MCD)**—an autosomal recessive genetic disorder characterized by a biotin deficiency. Symptoms include seizures, developmental delay, eczema, and hearing loss. Other symptoms are immune system impairment, skin rashes,

hair loss, and intellectual and developmental disability that are treatable with oral biotin supplements.

**Neonatal carnitine palmitoyl transferase deficiency-type II (CPT-II)**—an autosomal recessive genetic disorder of mitochondrial fatty acid oxidation that presents in three (3) forms. The classic form has adult onset of exercise-induced muscle weakness, often with rhabdomyolysis and myoglobinuria that may be associated with renal failure. A second form that is often fatal between three (3) and eighteen (18) months of age has symptoms of hepatomegaly, non-ketotic hypoglycemia, cardiomyopathy, hypotonia, and muscle weakness. A severe form presents in newborns with non-ketotic hypoglycemia, cardiomyopathy, hypotonia, muscle weakness, and renal dysgenesis in some patients.

**Newborn**—an infant under four (4) weeks of age.

**Phenylketonuria (PKU)**—the metabolic disease of the newborn in which metabolites of phenylalanine appear in the urine.

**Propionic acidemia (PROP)**—an autosomal recessive genetic disorder characterized by protein intolerance, vomiting, failure to thrive, lethargy, and profound metabolic acidosis. If not treated early, brain damage, coma, seizures and death can occur.

**Short chain acyl-CoA dehydrogenase deficiency (SCAD)**—an autosomal recessive genetic disorder of fatty acid beta oxidation with a usual clinical onset between the second month and second year of life, with some presenting within a few days of birth and some in adulthood. Symptoms include hypotonia, progressive muscle weakness, developmental delay, and seizures. Symptoms worsen with seemingly innocuous illness that may lead to lethargy, coma apnea, cardiopulmonary arrest, or sudden unexplained death.

**Short chain hydroxy acyl-CoA dehydrogenase deficiency (SCHAD)**—an autosomal recessive genetic disorder of mitochondrial fatty acid beta oxidation for which a complete spectrum of presentation has not been defined. Most patients have hypoglycemia as the major symptom along with seizures, neurologic sequela or death as the outcome. Several present in the first days or months of life with hypoglycemic seizures secondary to hyperinsulinism. Some patients present after one (1) year with acute onset of vomiting, lethargy, and hyponatremic seizures.

**Trifunctional protein deficiency (TFP)**—an autosomal recessive mitochondrial fatty acid oxidation genetic disorder characterized by an inability to break down long-chain fatty acids into an energy source. Metabolic crises can occur when fasting, as well as hypoglycemia, lethargy, hypotonia, myopathy, failure to thrive, cardiomyopathy, and neuropathy. Severe untreated cases may present as SIDS.

**Tyrosinemia type I (TYRO-I)**—an autosomal recessive genetic disorder that causes severe liver disease in infancy. Affected persons develop cirrhosis of the liver and eventually require liver transplantation. The most severe form causes symptoms within

the first months of life. These infants experience poor weight gain, enlarged liver and spleen, swelling of the legs, increased tendency of bleeding. Even with therapy death frequently occurs within six (6) to nine (9) months of life for those with the severe form. Children with a less severe form also suffer from enlargement of the liver, spleen, poor weight gain, vomiting, and diarrhea.

**Very long-chain acyl-CoA dehydrogenase deficiency (VLCAD)**—an autosomal recessive genetic disorder in which the body cannot oxidize fatty acids because of a missing or mal-functioning enzyme. Symptoms include hypoketotic hypoglycemia, hepatocellular disease, and cardiomyopathy. Fatal infantile encephalopathy may be the only indication of the condition.

**Subsection 2600.8 of Chapter 26 (Maternity Centers) is amended to read as follows:**

2600.8        Each maternity center shall, in addition to the other requirements of this chapter, comply with the requirements of sections 2100 and 2101 regarding newborn hearing screening and newborn testing for metabolic disorders.

## PUBLIC SERVICE COMMISSION OF THE DISTRICT OF COLUMBIA

NOTICE OF FINAL RULEMAKINGFormal Case No. 988, In the Matter of the Development of Universal Service Standards and a Universal Service Trust Fund for the District of Columbia

The Public Service Commission of the District of Columbia (“Commission”), pursuant to its authority under D.C. Code § 34-802 and D.C. Code § 34-2003 (2001 Ed.), hereby gives notice of the amendment of Chapter 28 of Title 15 DCMR. The Notice of Proposed Rulemaking to amend these rules was published in the *D.C. Register* on July 17, 2009 at *D.C. Reg. 5768-5770*. These amendments clarify the reimbursement process for eligible telecommunications carriers. The final version of these rules contains no modifications from the Notice of Proposed Rulemaking. Final action adopting these rules was taken September 22, 2009 by Commission Order No. 15552. The final rules listed below will become effective upon publication of this notice in the *D.C. Register*. Additional copies of the final rules may be obtained by writing Dorothy Wideman, Commission Secretary, Public Service Commission of the District of Columbia, 1333 H Street, NW, 2<sup>nd</sup> Floor, West Tower, Washington, DC 20005.

**CHAPTER 28 UNIVERSAL SERVICE****2803 DISTRICT OF COLUMBIA UNIVERSAL SERVICE TRUST FUND**

2803.1 Funds from the DC USTF will be used to support the enumerated services listed in 2802.1 as follows:

To reimburse eligible telecommunications carriers (“ETCs”) for the reasonable investments and expenses not recovered from the federal universal service low-income fund. The amount to be reimbursed shall be calculated for each ETC to be the remainder of the ETC’s retail tariffed rate less funding from the Federal Universal Service Low Income Fund less the tariffed lifeline rate for each eligible customer subscribing to the ETC’s lifeline service, not to exceed \$6.50 for each eligible customer. For ETCs that have a universal service program for low-income seniors, the amount to be reimbursed shall be similarly calculated, not to exceed \$8.50.

**2805 SIZING THE DISTRICT OF COLUMBIA UNIVERSAL SERVICE TRUST FUND**

2805.3 On or before November 30 of each year, the Commission shall establish a budget for the upcoming year after receipt of comments on the Fund Administrator’s proposed budget.

**2809 UNIVERSAL SERVICE FUND AUDIT**

2809.5 If the result of the audit reveals evidence of fraud or mismanagement, such results will be forwarded to the Office of the Inspector General and the District of Columbia Office of the Attorney General for further review.

**2813 REPORTING REQUIREMENTS FOR THE DC USTF ADMINISTRATOR**

2813.1 On a quarterly basis, the Fund Administrator shall submit to the Commission a report including:

- (a) A statement of collections and distributions from the universal service fund for each local exchange carrier;
- (b) A statement detailing the purpose for which the universal service funds were used (i.e. to support an enumerated service listed in § 2802.1 or for verification of lifeline eligibility); and
- (c) A record of total cost of universal service fund administration.

2813.2 On September 30 every year after the establishment of the DC USTF, the Fund Administrator shall submit to the Commission a report that includes a proposed budget for the upcoming year.

**2820 DISTRICT OF COLUMBIA LIFELINE SERVICE PROGRAM**

2820.5 When the entity responsible for certifying Lifeline customers notifies an ETC that a list of customers no longer qualifies for Lifeline service, and when the Commission orders termination of Lifeline service for those customers, the Lifeline rate for those customers will revert to the serving ETC's standard tariffed retail rate.

**2823 DEFINITIONS**

“Low-income senior” means a person aged 65 or older that qualifies for Lifeline service.

## PUBLIC SERVICE COMMISSION OF THE DISTRICT OF COLUMBIA

NOTICE OF FINAL RULEMAKINGFORMAL CASE NO. 977, IN THE MATTER OF THE INVESTIGATION INTO THE QUALITY OF SERVICE OF WASHINGTON GAS LIGHT COMPANY, DISTRICT OF COLUMBIA DIVISION, IN THE DISTRICT OF COLUMBIA

1. The Public Service Commission of the District of Columbia (“Commission”), pursuant to the D.C. Official Code, § 2-505 (2006 Repl.) and § 34-802 (2001 Ed.), hereby gives notice of final rulemaking action, adopting a new Chapter 37 of Title 15 of the District of Columbia Municipal Regulations (“DCMR”), commonly referred to as the “Natural Gas Quality of Service Standards” (“NGQSS”). The Commission issued a Notice of Proposed Rulemaking (“NOPR”) which was published in the *D.C. Register* on February 27, 2009, giving notice of the Commission’s intent to adopt Chapter 37 of Title 15 DCMR.<sup>1</sup> Comments were filed in response to the NOPR. However, after reviewing all comments, the Commission determined that further revisions were unwarranted.

2. As indicated in the NOPR, the regulations set forth standards to establish requirements for ensuring that natural gas utilities and natural gas service providers operating in the District of Columbia (“District”) meet an adequate level of quality and reliability in the natural gas service provided to District residents. The adoption of these new provisions will establish the rights and responsibilities of the consumer, the natural gas utility, and competitive energy suppliers. Accordingly, the Commission hereby adopts Chapter 37 of Title 15 DCMR governing the NGQSS as contained in the *D.C. Register* on February 27, 2009. The rules will become effective upon publication of this Notice of Final Rulemaking in the *D.C. Register*. Copies of the rules may be obtained by contacting Dorothy Wideman, Commission Secretary, Public Service Commission of the District of Columbia, 1333 H Street, NW, West Tower, Suite 200, Washington, DC 20005. Copies may also be obtained on the Commission’s website at [www.dcpsc.org](http://www.dcpsc.org).

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<sup>1</sup> 56 *D.C. Register* 1827-1851 (February 27, 2009). In an effort to propose comprehensive rules, the Commission previously published several NOPRs. See 54 *D.C. Register* 4489-4507 (May 11, 2007); 55 *D.C. Register* 0065-0087 (January 4, 2008); and 55 *D.C. Register* 9109-9131 (August 22, 2008). The Commission considered all comments received on various provisions throughout the process and hereby adopts final rules.