Training Coordinator Conference Call

DATE and TIME:	July 27, 2010	2:00 - 3:30 EDT
CALL IN NUMBER	1-888-808-6959	Code 6254120367
FACILITATOR:	Janice Miller, Room 335N	
NOTES TAKEN BY:	Haylie Smith	
LES Representation on the Call:	Bay	
("X" = LES participated on the call)	Big Bend	
	Central	
	Gold Coast	
	Gulf Central	
	North Beaches	
	North Central	
	North Dade	
	Northeastern	
	Southernmost Coast	
	Southwest	
	Space Coast	
	Treasure Coast	
	West Central	
	Western Panhandle	

TOPIC	PRESENTER	RELATED DOCUMENTS/REFERENCES	OUTCOME
Roll Call	Janice Miller		
Review of Notes	Janice Miller	Notes From Training Coordinators Call on May 18, 2010	
Update on the Development of Service Coordinator Training Modules	Terry Hoover		
Solicit Feedback on Tips From the Field	Terry Hoover		
Statewide Meeting	Haylie Smith		
Status Report on Major Training Projects	Janice Miller for Liza Smith		
Next Call			August 17 from 2:00 - 3:30 EDT (Same Conference Call Number and Code)

Charlie Crist
Governor

Ana M. Viamonte Ros, M.D., M.P.H.
State Surgeon General

July 22, 2010

Subject FY 09-10 Audits and Maintenance of Effort Funding

Dear Early Steps Contract Provider,

Prior to the state fiscal year (FY) 09-10, we notified you that we had evaluated the state funding awarded via our contract with you for Early Steps Services and that we had made the determination that the funding should be classified as state Maintenance of Effort (MOE), instead of State Financial Assistance. Subsequent to notifying you of this change, an amendment was processed with an effective date of July 1, 2009, formally making this change to the contract for FY 09-10.

Last year we received questions concerning how the FY 08-09 state funding should be treated for audit purposes and the decision was made that since the contract had not been amended until FY 09-10, the funding should be treated as State Financial Assistance for FY 08-09. We wanted to be proactive in providing audit guidance for the state MOE funding received in FY 09-10 and request that you share a copy of this letter with your independent auditor prior to the commencement of their audit to assist them in developing their audit plan.

For purposes of the FY 09-10 audit and each subsequent fiscal year hereafter – the MOE funding must be included and tested Office of Management and Budget (OMB) Circular A-133 for federal funding. We will be processing an amendment in the next few months to ensure that this requirement is clearly identified in the Financial and Compliance Audit Attachment, Exhibit 1 of each Early Steps contract.

MOE funds are General Revenue, identified by the Other Cost Accumulator (OCA) GR000 and Tobacco Trust Fund, OCA TOBNP on the payment reports you receive.

As always, the Early Steps State Office appreciates your assistance and for the quality work you do providing services to Florida's children. Please contact your Contract Manager with any questions concerning this matter.

Best Regards

LynnMarie Price
Bureau Chief
Early Steps

4052 Bald Cypress Way
Bin A06
Tallahassee, FL 32399-1701

Phone: (850) 245-4200 Fax: (850) 921-8138
http://www.cms-kids.com/EarlyStepshome.htm

Florida Early Learning & Developmental Standards

To register, please contact:
Whitney Davis at
whitney.davis@flaawi.com

Presented by
Pamela Phelps, Ph.D.
The Creative Center for
Childhood Research and
Training, Inc. (CCCRT)

Sponsored by The Agency for
Workforce Innovation • Office of Early Learning

The Tipping Point: Preventing TV, Furniture, and Appliance Tip-Over Deaths and Injuries

The U.S. Consumer Product Safety Commission (CPSC) estimates that in 2006 16,300 children 5 years old and younger were treated in U.S. hospitals emergency rooms because of injuries associated with TV, furniture, and appliance tip-overs. Additionally, between 2000 and 2006, CPSC staff received reports of 134 tip-over related deaths involving this same age group.

Typically, injuries and deaths occur when children climb onto, fall against or pull themselves up on television stands, shelves, bookcases, dressers, desks, chests, and appliances. In some cases, televisions placed on top of furniture tip over and cause a child to suffer traumatic and sometimes fatal injuries.

CPSC offers the following simple, low-cost safety tips to prevent tip-overs:

- Furniture should be stable on its own. For added security, anchor chests or dressers, TV stands, bookcases, and entertainment units to the floor or attach to a wall.
- Place TVs on a sturdy, low-rise base. Avoid flimsy shelves.
- Push the TV as far back as possible.
- Keep remote controls and other attractive items off the TV stand so kids won't be tempted to grab for them and risk knocking the TV over.
- Make sure free-standing ranges and stoves are installed with anti-tip brackets.

There are voluntary safety standards in place for TV stands/carts, chests, bureaus, and dressers, which require that the furniture passes a stability test. If a piece of furniture violates the appropriate standard, the product can be recalled. 5004-0909

Cómo Evitar las Muertes y las Lesiones Asociadas con la Caída de Televisores, Muebles y Electrodomésticos

La Comisión para la Seguridad de los Productos de Consumo de los Estados Unidos (CPSC) estima que 16,300 niños menores de 5 años fueron atendidos en salas de emergencias de hospitales en los Estados Unidos en el año 2006 debido a lesiones asociadas con la caída de televisores, muebles y electrodomésticos. Además, desde el 2000 hasta el 2006, el personal de la CPSC recibió reportes sobre 134 muertes de niños de la misma edad asociadas a la caída de estos objetos.

Típicamente, estas lesiones y muertes ocurren cuando los niños se trepan, se recuestan o se empujan sobre muebles para televisores, armarios, tocadores, escritorios, cómodas y electrodomésticos. En algunos casos los televisores colocados sobre muebles caen sobre los niños causándoles lesiones traumáticas y a veces fatales.

La CPSC ofrece los siguientes consejos de seguridad sencillos y económicos para evitar estos peligros:

- Verifique que los muebles son estables por sí solos. Para mayor seguridad, fije a la pared o ancle en el piso los tocadores, cómodas, muebles para televisores, armarios y unidades de entretenimiento.
- Coloque los televisores en muebles estables, de baja altura y apropiados para su tamaño. Empuje el televisor lo más lejos posible del frente del mueble.
- Retire de la mesa para el televisor artículos como el control remoto y juguetes que tienten al niño a treparse sobre el mueble para alcanzarlos.
- Asegúrese de que las estufas independientes sean instaladas con soportes para evitar volcamientos.

Existen normas voluntarias de seguridad para los muebles para televisores, las cómodas y los tocadores que requieren que éstos pasen una prueba de estabilidad. Si un mueble viola esta norma, el producto puede ser retirado del mercado.

5004-0909

NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD

Early Experiences Can Alter Gene Expression and Affect Long-Term Development

WORKING PAPER 10

Center on the Developing Child HARVARD UNIVERSITY

NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD

MEMBERS

Jack P. Shonkoff, M.D., Chair
Julius B. Richmond FAMRI Professor of Child Health and Development, Harvard School of Public Health and Harvard Graduate School of Education; Professor of Pediatrics, Harvard Medical School and Children's Hospital Boston; Director, Center on the Developing Child, Harvard University

Pat Levitt, Ph.D., Science Director
Director, Zilkha Neurogenetic Institute; Provost Professor of Neuroscience, Psychiatry & Pharmacy; Chair, Department of Cell and Neurobiology, Keck School of Medicine, University of Southern California

W. Thomas Boyce, M.D.
Sunny Hill Health Centre/BC Leadership Chair in Child Development; Professor, Graduate Studies and Medicine, University of British Columbia, Vancouver

Judy Cameron, Ph.D.
Professor of Psychiatry, University of Pittsburgh

Greg J. Duncan, Ph.D.
Distinguished Professor, Department of Education, University of California, Irvine

Nathan A. Fox, Ph.D.
Distinguished University Professor; Director, Child Development Laboratory, University of Maryland College Park

Megan Gunnar, Ph.D.
Regents Professor and Distinguished McKnight University Professor, Institute of Child Development, University of Minnesota

Linda C. Mayes, M.D.
Arnold Gesell Professor of Child Psychiatry, Pediatrics, and Psychology, Yale Child Study Center;
Special Advisor to the Dean, Yale School of Medicine

Bruce S. McEwen, Ph.D.
Alfred E. Mirsky Professor; Head, Harold and Margaret Milliken Hatch Laboratory of Neuroendocrinology, The Rockefeller University

Charles A. Nelson III, Ph.D.
Richard David Scott Chair in Pediatric Developmental Medicine Research, Children's Hospital Boston; Professor of Pediatrics and Neuroscience, Harvard Medical School

Ross Thompson, Ph.D.
Professor of Psychology, University of California, Davis

CONTRIBUTING MEMBERS

Susan Nall Bales
President, FrameWorks Institute

Philip A. Fisher, Ph.D.
Professor of Psychology, University of Oregon
Senior Research Scientist, Oregon Social Learning Center & Center for Research to Practice

William Greenough, Ph.D.
Swanlund Professor of Psychology, Psychiatry, and Cell and Developmental Biology; Director, Center for Advanced Study at University of Illinois, Urbana-Champaign

Eric Knudsen, Ph.D.
Edward C. and Amy H. Sewall Professor of Neurobiology, Stanford University School of Medicine

Deborah Phillips, Ph.D.
Professor of Psychology and Associated Faculty, Public Policy Institute; Co-Director, Research Center on Children in the U.S., Georgetown University

Arthur J. Rolnick, Ph.D.
Senior Vice President and Director of Research, Federal Reserve Bank of Minneapolis

PARTNERS

The FrameWorks Institute
The National Governors Association Center for Best Practices
The National Conference of State Legislatures

SPONSORS

The Birth to Five Policy Alliance
The Buffett Early Childhood Fund
The Norlien Foundation

About the Authors

The National Scientific Council on the Developing Child, housed at the Center on the Developing Child at Harvard University, is a multi-disciplinary collaboration designed to bring the science of early childhood and early brain development to bear on public decision-making. Established in 2003, the Council is committed to an evidence-based approach to building broad-based public will that transcends political partisanship and recognizes the complementary responsibilities of family, community, workplace, and government to promote the well-being of all young children. For more information, go to www.developingchild.net.

Please note: The content of this paper is the sole responsibility of the Council and does not necessarily represent the opinions of the funders or partners.

Suggested citation: National Scientific Council on the Developing Child (2010). *Early Experiences Can Alter Gene Expression and Affect Long-Term Development: Working Paper No. 10*. http://www.developingchild.net

© May 2010, National Scientific Council on the Developing Child, Center on the Developing Child at Harvard University

FIRST PRINTING: MAY 2010

The Issue

NEW SCIENTIFIC RESEARCH SHOWS THAT ENVIRONMENTAL INFLUENCES CAN ACTUALLY AFFECT whether and how genes are expressed. Thus, the old ideas that genes are "set in stone" or that they alone determine development have been disproven. In fact, scientists have discovered that early experiences can determine how genes are turned on and off and even whether some are expressed at all.[1,2,3] Therefore, the experiences children have early in life—and the environments in which they have them—shape their developing brain architecture and strongly affect whether they grow up to be healthy, productive members of society. This growing scientific evidence supports the need for society to re-examine the way it thinks about the circumstances and experiences to which young children are exposed.

The approximately 23,000 genes that children inherit from their parents form what is called the "structural genome." Scientists liken the structural genome to the hardware of a computer—both determine the boundaries of what's possible, but neither works without an operating system to tell it what to do. In the genome, that operating system is called the epigenome.[4] Like the software in an operating system, the epigenome determines which functions the genetic "hardware" does and does not perform.[5] This system is built over time as positive experiences, such as exposure to rich learning opportunities, or negative influences, such as environmental toxins or stressful life circumstances, leave a chemical "signature" on the genes. These signatures can be temporary or permanent, and both types affect how easily the genes are switched on or off. For example, even though identical twins have the same structural genomes, their different experiences result in different epigenomes.[6] These differing experiences leave signatures on the epigenome that cause some genes to be expressed differently. This explains why genetically identical twins, though similar in many ways, can exhibit different behaviors, skills, health, and achievement in both school and, later, in the workplace.

The field of epigenetics is relatively new and at the cutting-edge of the biological sciences. To date, scientists have found that *temporary* epigenetic chemical modifications control when and where most of our genes are turned on and off. This, however, is not the entire story. Certain experiences can also cause *enduring* epigenetic modifications in hundreds of genes that have already been identified, and the list is growing.[7,8] Increasing evidence shows that experience-driven, chemical modifications of these latter genes appear to play particularly key roles in brain and behavioral development. This new knowledge has motivated scientists to look more closely at the factors that shape the epigenome and to study whether interventions can reverse these modifications when negative changes occur.

Nutritional status, exposure to toxins and drugs, and the experiences of interacting with varied environments can all modify an individual's epigenome.[9] Epigenetic instructions that change how and when certain genes are turned on or off can cause temporary or enduring health problems. Moreover, research in both animals and humans shows that some epigenetic changes that occur in the fetus during pregnancy can be passed on to later generations, affecting the health and welfare of children, grandchildren, and their descendents.[10,11,12] For example, turning on genes that increase cell growth, while at the same time switching off genes that suppress cell growth, has been shown to cause cancer.[13,14] Repetitive, highly stressful experiences can cause epigenetic changes that damage the systems that manage one's response to adversity later in life.[2,3,15] On the other hand, supportive environments and rich learning experiences generate positive epigenetic

Like the software in a computer's operating system, the epigenome determines which functions the genetic "hardware" does and does not perform.

signatures that *activate* genetic potential.[16] In this second case, the stimulation that occurs in the brain through active use of learning and memory circuits can result in epigenetic changes that establish a foundation for more effective learning capacities in the future.[17,18]

As we get older, new experiences can continue to change our epigenome. However, science tells us that the chemical signatures imprinted on our genes during fetal and infant development can have significant influences on brain architecture that last a lifetime. Stated simply, the discovery of the epigenome provides an explanation, at the molecular level, for why and how early positive and negative experiences can have lifelong impacts.[2,3,19,20]

Policymakers can use this knowledge to inform decisions about the allocation of resources for interventions that affect the life circumstances of young children—knowing that effective interventions can literally alter how children's genes work and, thereby, have long-lasting effects on their mental and physical health, learning, and behavior. In this respect, the epigenome is the crucial link between the external environments that shape our experiences and the genes that guide our development.

What Science Tells Us

OVER THE PAST 50 YEARS, EXTENSIVE RESEARCH has demonstrated that the healthy development of all organs, including the brain, depends on how much and when certain genes are expressed. When scientists say that genes are "expressed," they are referring to whether they are turned on or off—essentially whether and when genes are activated to do certain tasks. Research has shown that there are many non-inherited environmental factors and experiences that have the power to chemically mark genes and control their functions. These influences create a new genetic landscape, which scientists call the epigenome. Some of these experiences lead to chemical modifications that change the expression of genes temporarily, while increasing numbers have been discovered that leave chemical signatures that result in an enduring change in gene expression.

Early prenatal or postnatal experiences and exposures influence long-term outcomes by chemically altering the structure of genes. Known as *epigenetic* modification (from the Greek root *epi*, meaning upon or over), these chemical signatures are written on top of the gene without actually altering the genetic code itself.[21] Instead, the signatures attract or repel other chemicals that help the genes produce the proteins that are the building blocks our brains and bodies need to develop. Research tells us that some genes can only be modified epigenetically during certain periods of development, defined as *critical periods* of modification, while other genes are open to alterations throughout life.[2,3,22,23,24,25]

Epigenetic modification typically occurs in cells that comprise organ systems, thereby influencing how these structures develop and function. Experiences that change the epigenome early in life, when the specialized cells of organs such as the brain, heart, or kidneys are first developing, can have a powerful impact on physical and mental health for a lifetime.[26] We are also learning from new scientific discoveries in both animals and humans that environmental factors, such as certain drugs or the nutritional status of the mother, have the potential to cause epigenetic changes to genes in egg or sperm cells in the fetus. When such changes occur, this new chemical signature of the DNA is enduring and can be inherited by future generations.[27,28,29]

The brain is particularly responsive to experiences and environments during early development, which influences how well or poorly its architecture matures and functions. We know from extensive research that the physiological activity created by experience is powerful in shaping brain architecture and actually changes the chemistry that encodes the genes in brain cells.[30] Put simply, the brain adapts to the experiences it has. Certain types of adaptations result in healthy systems, such as effective learning and memory, and other adaptations lead to the development of unhealthy systems, such as setting a stress response activation level too high

or too low. The physiological activity caused by positive mastery experiences can lead to epigenetic changes that control the expression of genes in brain cells that are essential for successful learning.[17,18,31] In a parallel fashion, exposure to damaging levels of stress early in life can lead to long-lasting epigenetic changes in brain cells that direct how our bodies respond to adversity throughout the lifespan.[32] In short, early experiences cause epigenetic adaptations in the brain that influence whether, when, and how genes build the capacity for future skills to develop.

Modification of the epigenome caused by stress during fetal and child development affects how well or poorly we respond to stress as adults and can result in increased risk of adult disease. Some of our genes provide instructions for how our bodies respond to stress, and research has shown that these genes are clearly subject to epigenetic modification. For example, research in animals has shown that stressful experiences to which the pregnant mother is exposed, or to which the offspring is exposed soon after birth, can produce epigenetic changes that chemically modify the receptor in the brain that controls the stress hormone cortisol and, therefore, determines the body's response to threat (the fight-or-flight response).[19,33,34] Healthy stress responses are characterized by an elevation in blood cortisol followed by a return to baseline to avoid a highly activated state for a prolonged period of time. If young children or pregnant mothers experience toxic stress—as a result of serious adversity (such as chronic neglect, abuse, or exposure to violence) in the absence of protective relationships—persistent epigenetic changes can result.[32] These modifications have been shown to cause prolonged stress responses, which can be likened to revving a car engine for long periods of time. Excessive stress has been correlated with changes in brain architecture and chemistry as well as animal behaviors that resemble anxiety and depression in

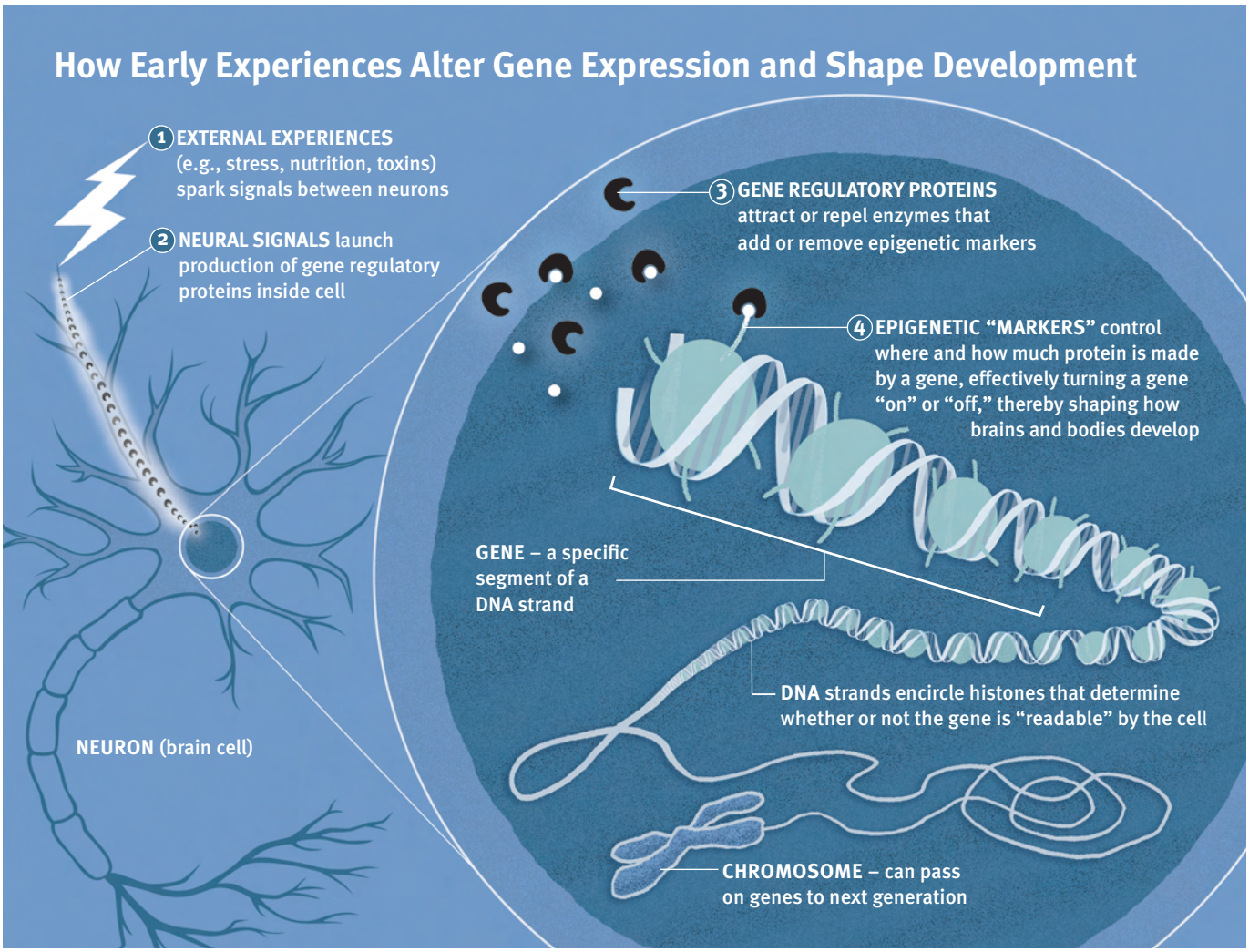

ILLUSTRATION BY BETSY HAYES

humans.[35,36,37,38,39,40] Human studies have found connections between highly stressful experiences in children and increased risk for later mental illnesses, including generalized anxiety disorder and major depressive disorder.[41,42,43] Atypical stress responses over a lifetime can also result in increased risk for physical ailments, such as asthma, hypertension, heart disease and diabetes.[29,32,41,42,43,44,45,46,47,48]

In addition to adverse experiences, a wide variety of chemicals, nutrients, and drugs are also capable of modifying the epigenome for long-lasting effects on gene expression. Epigenetic modification caused by exposure to toxic substances can either turn genes off or on, and both conditions have been linked to increased risk for mental and physical illnesses. Certain dietary supplements (such as excessive amounts of folic acid, choline, or vitamin B_{12}) and chemicals (such as bisphenol A, or BPA, which is found in some plasticware) can turn genes off.[1,8,49,50] Some genes that can be turned on by heavy metals, such as cadmium, nickel, and lead, have been linked to the cellular over-activity that results in an increased risk for certain kinds of cancer.[28,51,52] Many organs, including the brain, are most vulnerable to the influence of these substances on gene expression during the period of fetal and infant development, when basic organ systems are being built. These resulting differences in gene expression, in turn, can lead to fundamental changes in brain architecture and the biological systems that govern how well we function later in life.

Recent research demonstrates that even after the epigenome has been modified, there may be ways to alter it again that actually can reverse negative changes and restore functioning. Experiments in animals have shown that certain types of epigenetic modifications that were thought to be permanent can be reversed under certain conditions.[35,53] Most recently, researchers found that stressful experiences during early postnatal development resulted in epigenetic modification that caused exaggerated stress responses in adult animals, and that subsequent drug treatment of the adults could eliminate these adverse DNA changes. In this case, reversing the chemical modification resulted in increased expression of genes that control the stress response, and when exposed to subsequent stress, the treated adult animals had a normal response (that is, the adverse effect of the early postnatal experience had, indeed, been reversed). We now know that the same types of epigenetic chemical modifications can occur in adult humans who endured extreme stress as children, such as from physical abuse.[19] This possibility of reversibility is generating a flurry of research activity, because it has direct implications for developing new interventions for physical and mental illnesses that we now know are due in part to epigenetic modification. As promising as this work appears to be, further research is needed to determine if and how the reversal of epigenetic modifications can be achieved in humans.

Correcting Popular Misrepresentations of Science

UNTIL RECENTLY, THE INFLUENCES OF GENES were thought to be set, and the effects of children's experiences and environments on brain architecture and long-term physical and mental health outcomes remained a mystery. That lack of understanding led to several misleading conclusions about the degree to which negative and positive environmental factors and experiences can affect the developing fetus and young child. The following misconceptions are particularly important to set straight.

Contrary to popular belief, the genes inherited from one's parents do not set a child's future development in stone. Variations in DNA sequences between individuals certainly influence the way in which genes are expressed and how the proteins encoded by those genes will function. But that is only part of the story—the environment in which one develops, before and soon after birth, provides powerful experiences that chemically modify certain genes which, in turn, define how much and when they are expressed. Thus, while genetic factors exert

potent influences, environmental factors have the ability to alter family inheritance.

Although frequently misunderstood, adverse fetal and early childhood experiences can—and do—lead to physical and chemical changes in the brain that can last a lifetime. Injurious experiences, such as malnutrition, exposure to chemical toxins or drugs, and toxic stress before birth or in early childhood are not "forgotten," but rather are built into the architecture of the developing brain through the epigenome. The "biological memories" associated with these epigenetic changes can affect multiple organ systems and increase the risk not only for poor physical and mental health outcomes but also for impairments in future learning capacity and behavior.

Despite some marketing claims to the contrary, the ability of so-called enrichment programs to enhance otherwise healthy brain development is not known. While parents and policymakers might hope that playing Mozart recordings to newborns will produce epigenetic changes that enhance cognitive development, there is absolutely no scientific evidence that such exposure will shape the epigenome or enhance brain function. What research *has* shown is that specific epigenetic modifications do occur in brain cells as cognitive skills like learning and memory develop and that repeated activation of brain circuits dedicated to learning and memory through interaction with the environment, such as reciprocal "serve and return" interaction with adults,[54] facilitates these positive epigenetic modifications. We also know that sound maternal and fetal nutrition, combined with positive social-emotional support of children through their family and community environments, will reduce the likelihood of negative epigenetic modifications that increase the risk of later physical and mental health impairments.

> **Adverse fetal and early childhood experiences can—and do—lead to physical and chemical changes in the brain that can last a lifetime.**

The Science-Policy Gap

THE FACT THAT THE GENOME IS VULNERABLE TO modification by toxic stress, nutritional problems, and other negative influences underscores the importance of providing supportive and nurturing experiences for young children in the earliest years, when brain development is most rapid. From a policy perspective, it is in society's interest to strengthen the foundations of healthy brain architecture in all young children to maximize the return on future investments in education, health, and workforce development. In this context, the epigenome is the chemical signature that explains how early life experiences become embedded in the circuitry of the developing brain and are associated with lifelong consequences. Research now shows that interaction between adverse environments and the genes we inherit—through the epigenome—can increase the risk for long-term negative mental and physical health outcomes. Nevertheless, many policy decisions do not yet reflect this growing knowledge, which logically calls for reducing the exposure of pregnant women and young children to environments and experiences that can have significant negative effects on the epigenome (and therefore powerful, indirect influences on genes). This gap between what we know and what we do is illustrated by the following four examples:

Child welfare. Because threatening or harmful environments can produce epigenetic modifications that affect a child adversely for a lifetime, the management of child abuse or neglect cases by child protective services is extremely time-sensitive. This sense of urgency is particularly important with respect to decisions about custody arrangements that involve non-family placements. The biology of adversity also tells us that assessment and planning for the mental health and broad developmental needs of vulnerable children—with the assurance of

nurturing, protective, and stable relationships as the highest priority—require attention in the child welfare system comparable to the conventional focus placed primarily on physical safety.

Mandated maternal employment and public assistance. Whether low-income mothers with very young children should be required to work in order to be eligible for public assistance is a political decision. Policies informed by scientific knowledge about early brain development and epigenetics, however, would link mandated maternal employment to a parallel investment in high-quality early care and education programs for affected children. When policies view child care simply as a custodial service whose primary purpose is to facilitate maternal work outside the home, they reflect a lack of understanding of extensive scientific evidence about how the developing architecture of the brain is shaped by epigenetic influences associated with the quality of adult-child interactions in the early childhood years. In contrast, policies that view high-quality child care and early education programs as strategic interventions to improve the life prospects of children whose parents have limited education and low income are more likely to increase the prosperity of communities across generations.

Prenatal and newborn health care. The fetal period is a highly active time for organ development and epigenetic modification, yet investment in prenatal services remains uneven. Policies that assure the availability, accessibility, and affordability of individualized support and monitoring of all pregnancies create a safety net that prevents harm and detects problems at a point when appropriate interventions can reduce the negative consequences of toxic stress and other adverse environmental exposures.

Support for new parents. The United States is one of very few developed nations that does not provide some amount of paid family leave for all parents after the birth or adoption of a baby. For parents of newborns who do not have the economic resources to make ends meet in the absence of paid employment, the supportive relationships that promote positive epigenetic changes and help very young children to manage stress can be compromised by a premature return to work and the inability to secure high-quality care for a young infant. Family leave is one way of helping parents build these critical relationships, but other policies that support parents during this important transition time can also have important, short-term effects on the quality of family life as well as long-term impacts that bring high returns to all of society.

Implications for Policy and Programs

BECAUSE EARLY EXPERIENCES CAN ALTER THE epigenome and influence developing brain architecture, policies affecting the life circumstances of pregnant women and young children can have enormous implications for all of society. The varied effects of environments on the epigenome are evident from the time of early embryonic development and extend into the early childhood years. Science tells us that children can be helped to reach their full potential through both appropriate experiences in the earliest years and the reduction of sources of toxic stress that can alter the epigenome and increase the risk of long-term problems in physical and mental health. Thus, public policies that harness the basic principles of neuroscience and epigenetics to address the needs of young children are likely to also generate long-term benefits, such as healthier communities and a more prosperous society.

The documented effects of toxic stress on negative epigenetic adaptations demonstrate the urgent need to alleviate sources of significant adversity as early as possible in the lives of children who live in threatening environments. In order to be maximally effective, programs and services targeting the precipitants of excessive stress—such as child abuse and neglect, violent neighborhoods and families, and caregiver mental illness—must have prompt access to specific expertise in the target areas in order to move quickly to protect young children from epigenetic changes that can lead to lifelong problems

in learning, behavior, and health. Because the clock is always ticking when the basic architecture of the brain is developing, informed policymakers understand that a delayed response to the needs of young children who are experiencing significant adversity jeopardizes their individual well-being as well as the broader human capital needs of society.

Epigenetic changes caused by the exposure of pregnant women, infants, and toddlers to environmental toxins, prescription drugs, alcohol, and illicit substances require an urgent look at what safeguards can be implemented to prevent such exposures. Lead paint laws are a good example of public policy that has been successful in reducing the harmful consequences of environmental toxins. Less aggressive policies with respect to mercury and organophosphate insecticides, on the other hand, are just two examples of many missed opportunities to mitigate the well-documented, adverse effects of environmental hazards on pregnant women and young children. The serious and continuing impact of prenatal exposure to alcohol and a wide variety of chemical substances (including prescription drugs) on child health and development calls for a more vigorous approach to environmental policies and public education. In view of the well-established scientific fact that embryonic, fetal, and early childhood brain development is considerably more susceptible to damage from neurotoxins than the mature brain of an adult, the establishment of safe levels of exposure to toxic substances should be based on scientific data that recognize the critical link between vulnerability and age, and that focus primarily on the best information available for the youngest children.[55]

Because prenatal and early postnatal experiences can affect long-term outcomes through epigenetic influences, the provision of high-quality health services and nutritional support for all pregnant women, infants, and toddlers would be likely to reduce preventable diseases across the lifespan—as well as the costly treatments for them. A range of currently available programs and systems has been established to meet these needs. Assuring access to appropriate, affordable, high-quality services that are well implemented, however, remains an elusive goal, particularly for many of the most disadvantaged families and those whose primary language is not English. Policies that succeed in connecting all pregnant women and young children to medical and nutrition services that match their individualized needs will produce measurable benefits in population health.

Certain epigenetic changes in humans can be transferred across generations, thereby underscoring important, long-term implications for policies that affect the circumstances in which young children are raised. Effective policies and programs that address conditions associated with economic hardship with or without other sources of adversity—especially those targeted to help families during pregnancy—can not only improve birth outcomes and short-term conditions for young children but should also be viewed as investments in building a stronger foundation for healthy communities and future prosperity across generations. When policymakers support positive environments for pregnant women and very young children, they reduce the risk of intergenerational transfer of negative epigenetic changes that can lead to impaired health, diminished learning capacity, and poor parenting of the next generation.

Because discoveries about the epigenome and its lifelong effects are so recent, a multi-faceted education campaign could bring important new information to a wide range of important audiences, including health professionals, judges and lawyers, educators, caregivers, families, and the general public. A broad-based understanding of cutting-edge, developmental science by people who influence the experiences and environments of young children could provide a framework for greater alignment and integration of the current patchwork of services and supports provided to families. In this vein, greater understanding of how toxic stress, poor nutrition, and toxic chemical and drug exposures can increase lifelong risks for physical and mental health impairments by changing the chemistry of our children's DNA would provide a powerful foundation for more effective public action to address the needs of young children—and all of society—for generations to come.

References

1. Meaney, M. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, *81(1)*, 41–79.
2. Szyf, M. (2009a). Early life, the epigenome and human health. *Acta Paediatrica, 98*(7), 1082-1084.
3. Szyf, M. (2009b). The early life environment and the epigenome. *Biochimica Biophysica Acta (BBA), 1790*(9), 878-885.
4. Waddington, C. H. (1942). Canalization of development and the inheritance of acquired characters. *Nature, 150,* 563-565.
5. Dolinoy, D. C., Weidman, J. R., & Jirtle, R. L. (2007). Epigenetic gene regulation: Linking early developmental environment to adult disease. *Reproductive Toxicology, 23*(3): 297-307.
6. Kaminsky, Z. A., Tang, T., Wang, S., Ptak, C., Oh, G. H. T., Wong, A. H. … & Petronis, A.. (2009). DNA methylation profiles in monozygotic and dizygotic twins. *Nature Genetics, 42,* 240-245.
7. Crews, D. (2008). Epigenetics and its implications for behavioral neuroendocrinology. *Frontiers in Neuroendocrinology, 29*(3), 344-357.
8. Dolinoy, D. C. & Jirtle, R. L. (2008). Environmental epigenomics in human health and disease. *Environmental and Molecular Mutagenesis, 49*(1), 4-8.
9. Bernstein, B. E., Meissner, A., & Lander, E. S. (2007). The mammalian epigenome. *Cell*, 128(4), 669-81.
10. Anway, M. D., Cupp, A. S., Uzumcu, M., & Skinner, M. K. (2005). Epigenetic transgenerational actions of endocrine disruptors and male fertility. *Science 308*, 1466-1469.
11. Champagne, F. A. (2010). Epigenetic influences of social experiences across the lifespan. *Developmental Psychobiology,* 1-13.
12. Newbold, R. R., Padilla-Banks, E., & Jefferson, W. N. (2006) Adverse effects of the model environmental estrogen diethylstilbestrol are transmitted to subsequent generations. *Endocrinology, 146,* S11-S17.
13. Smith, L. T., Otterson, G. A., & Plass, C. (2007). Unraveling the epigenetic code of cancer for therapy. *Trends in Genetics, 23*(9), 449-456.
14. Giacinti, L., Vici, P., & Lopez, M. (2008). Epigenome: A new target in cancer therapy. *Clinica Terapeutica, 159*(5), 347-360.
15. Bagot, R. C., van Hasselt, F. N. , Champagne, D. L., Meaney, M. J., Krugers, H. J., & Joels, M. (2009). Maternal care determines rapid effects of stress mediators on synaptic plasticity in adult rat hippocampal dentate gyrus. *Neurobiology of Learning and Memory, 92*(3), 292-300.
16. Curley, J. P. (2009). Social enrichment during postnatal development induces transgenerational effects on emotional and reproductive behavior in mice. *Frontiers in Behavioral Neuroscience, 3,* 1-14.
17. Sweatt, J. D. (2007). An atomic switch for memory. *Cell, 129*(1), 23-4.
18. Sweatt, J. D. (2009). Experience-dependent epigenetic modifications in the central nervous system. *Biological Psychiatry, 65*(3), 191-7.
19. McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonte, B., Szyf, M. … & Meaney, M. J. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience, 12*(3), 342-348.
20. Roth, T. L., Lubin, F. D., Funk A., & Sweatt, J. (2009). Lasting epigenetic influence of early-life adversity on the BDNF gene. *Biological Psychiatry, 65*(9), 760-769.
21. Mellor, J., Dudek, P., & Clynes, D. (2008). A glimpse into the epigenetic landscape of gene regulation. *Current Opinion in Genetics & Development, 18*(2), 116-122.
22. Isles, A. R. & Wilkinson, L. S. (2008). Epigenetics: What is it and why is it important to mental disease? *British Medical Bulletin, 85*(1), 35-45.
23. Jirtle, R. L. (2008). Randy L. Jirtle, PhD: Epigenetics a window on gene dysregulation, disease. Interview by Bridget M. Kuehn. *Journal of the American Medical Association (JAMA), 299*(11): 1249-1250.
24. Nafee, T. M., Farrell, W. E., Carroll, W. D., Fryer, A. A., & Ismail, K. M. (2008). Epigenetic control of fetal gene expression. *BJOG: An International Journal of Obstetrics & Gynaecology, 115*(2), 158-168.
25. Sinclair, D. A. & Oberdoerffer, P. (2009). The ageing epigenome: Damaged beyond repair? *Ageing Research Reviews, 8*(3), 189-198.
26. Das, R., Hampton, D. D., & Jirtle, R. L. (2009). Imprinting evolution and human health. *Mammalian Genome, 20*(9-10), 563.
27. Bocock, P. N. & Aagaard-Tillery, K. M. (2009). Animal models of epigenetic inheritance. *Seminars in Reproductive Medicine, 27(5),* 369-79.
28. Suter, M. A. & Aagaard-Tillery, K. M. (2009). Environmental influences on epigenetic profiles. *Seminars in Reproductive Medicine, 27*(5), 380-390.
29. Swanson, J. M., Entringer, S. , Buss, C., & Wadhwa, P. D. (2009). Developmental origins of health and disease: Environmental exposures. *Seminars in Reproductive Medicine, 27*(5), 391-402.
30. Levitt, P. (2003). Structural and functional maturation of the developing primate brain. *Journal of Pediatrics,* 143(4), 35-45.
31. Miller, C. A., Campbell, S. L., & Sweatt, J. D. (2008). DNA methylation and histone acetylation work in concert to regulate memory formation and synaptic plasticity. *Neurobiology of Learning and Memory, 89*(4), 599-603.
32. Shonkoff, J. P., Boyce, W. T. , & McEwen, B S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA, 301*(21), 2252-2259.
33. Meaney, M. J., Szyf, M., & Seckl, J. R. (2007). Epigenetic mechanisms of perinatal programming of hypothalamic-pituitary-adrenal function and health. *Trends in Molecular Medicine, 13*(7), 269-277.
34. Weaver, I. C. G., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., & Meaney, M. J. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience, 7*(8), 847-854.

35. Champagne, F. A. & Curley, J. P. (2009). Epigenetic mechanisms mediating the long-term effects of maternal care on development. *Neuroscience and Biobehavioral Reviews, 33*(4), 593-600.
36. Champagne, F. A., Weaver, I. C., Diorio, J., Dymov, S., Szyf, M., & Meaney, M. J. (2006). Maternal care associated with methylation of the estrogen receptor-alpha1b promoter and estrogen receptor-alpha expression in the medial preoptic area of female offspring. *Endocrinology, 147*(6), 2909-2915.
37. Chen, Y., Dube, C. M., Rice, C. J., & Baram, T. Z. (2008). Rapid loss of dendritic spines after stress involves derangement of spine dynamics by corticotropin-releasing hormone. *The Journal of Neuroscience, 28*(11), 2903-2911.
38. Moriceau, S. & Sullivan, R. M. (2006). Maternal presence serves as a switch between learning fear and attraction in infancy. *Nature Neuroscience, 9,* 1004-1006.
39. Rice, C. J., Sandman, C. A., Lenjavi, M. R., & Baram, T. Z. (2008). A novel mouse model for acute and long-lasting consequences of early life stress. *Endocrinology, 149*(10), 4892-4900.
40. Thompson, J. V., Sullivan, R. M., & Wilson, D. A. (2008). Developmental emergence of fear learning corresponds with changes in amygdala synaptic plasticity. *Brain Research, 1200,* 58-65.
41. Bradley, R. G., Binder, E. B., Epstein, M. P., Tang, Y., Nair, H. P., Liu, W. ... & Ressler, K. J. (2008). Influence of child abuse on adult depression: Moderation by the corticotropin-releasing hormone receptor gene. *Archives of General Psychiatry, 65*(2), 190-200.
42. Gillespie, C. F., Bradley, B., Mercer, K., Smith, A., Conneely, K., Gapen, M. … & Ressler, K. (2009). Trauma exposure and stress-related disorders in inner city primary care patients. *General Hospital Psychiatry, 31*(6), 505-514.
43. Hovens, J. G., Wiersma, J. E., Giltay, E. J., van Oppen, P., Spinhoven, P., Penninx, B. W. & Zitman, F. G. (2009). Childhood life events and childhood trauma in adult patients with depressive, anxiety and comorbid disorders vs. controls. *Acta Psychiatrica Scandinavica.* Oct 30 [epub]
44. Jovanovic, T., Blanding, N. Q., Norrholm, S. D., Duncan, E., Bradley, B., & Ressler, K. J. (2009). Childhood abuse is associated with increased startle reactivity in adulthood. *Depression and Anxiety, 26*(11), 1018-1026.
45. Krupanidhi, S., Sedimbi, S. K., Vaishnav, G., Madhukar, S. S., & Sanjeevi, C. B. (2009). Diabetes-role of epigenetics, genetics, and physiological factors. *Zhong Nan Da Xue Xue Bao Yi Xue Ban, 34*(9), 837-845.
46. Quas, J. A., Carrick, N., Alkon, A., Goldstein, L., & Boyce, W. T. (2006). Children's memory for a mild stressor: The role of sympathetic activation and parasympathetic withdrawal. *Developmental Psychobiology, 48*(8), 686-702.
47. Weidman, J. R., Dolinoy, D. C., Murphy, S. K., & Jirtle, R. L. (2007). Cancer susceptibility: Epigenetic manifestation of environmental exposures. *Cancer Journal, 13,*(1), 9-16.
48. Wilson, A. G. (2008). Epigenetic regulation of gene expression in the inflammatory response and relevance to common diseases. *Journal of Periodontology, 79*(8): 1514-1519.
49. Dolinoy, D. C., Huang, D., & Jirtle, R. L. (2007). Maternal nutrient supplementation counteracts bisphenol A-induced DNA hypomethylation in early development. *Proceedings of the National Academy of Sciences of the United States of America, 104*(32), 13056-13061.
50. Waterland, R. A. & Jirtle, R. L. (2004). Early nutrition, epigenetic changes at transposons and imprinted genes, and enhanced susceptibility to adult chronic diseases. *Nutrition*, 20(1), 63-68.
51. Waisberg, M., Joseph, P., Hale, B., & Beyersmann, D. (2003). Molecular and cellular mechanisms of cadmium carcinogenesis. *Toxicology, 192*(2-3), 95-117.
52. Salnikow, K. & Zhitkovich, A. (2008). Genetic and epigenetic mechanisms in metal carcinogenesis and cocarcinogenesis: nickel, arsenic and chromium. *Chem. Res. Toxicol, 21*, 28-44.
53. Szyf, M. (2009c). Epigenetics, DNA methylation, and chromatin modifying drugs. *Annual Review of Pharmacology & Toxicology, 49,* 243-263.
54. National Scientific Council on the Developing Child (2007). The timing and quality of early experiences combine to shape brain architecture: Working paper no. 5. http://www.developingchild.net
55. National Scientific Council on the Developing Child (2006). Early exposure to toxic substances damages brain architecture: Working paper no. 4. http://www.developingchild.net

WORKING PAPER SERIES

Working Paper #1
Young Children Develop in an Environment of Relationships (2004)

Working Paper #2
Children's Emotional Development is Built into the Architecture of their Brains (2004)

Working Paper #3
Excessive Stress Disrupts the Architecture of the Developing Brain (2005)

Working Paper #4
Early Exposure to Toxic Substances Damages Brain Architecture (2006)

Working Paper #5
The Timing and Quality of Early Experiences Combine to Shape Brain Architecture (2007)

Working Paper #6
Mental Health Problems in Early Childhood Can Impair Learning and Behavior for Life (2008)

Working Paper #7
Workforce Development, Welfare Reform, and Child Well-Being (2008)

Working Paper #8
Maternal Depression Can Undermine the Development of Young Children (2009)

Working Paper #9
*Persistent Fear and Anxiety Can Affect Young Children's Learning and Development (*2010)

ALSO FROM THE CENTER ON THE DEVELOPING CHILD

A Science-Based Framework for Early Childhood Policy: Using Evidence to Improve Outcomes in Learning, Behavior, and Health for Vulnerable Children (2007)

The Science of Early Childhood Development: Closing the Gap Between What We Know and What We Do (2007)

Early Childhood Program Evaluations: A Decision-Maker's Guide (2007)

http://developingchild.harvard.edu/library/reports_and_working_papers/

NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD
Center on the Developing Child HARVARD UNIVERSITY

50 Church Street, 4th Floor, Cambridge, MA 02138
617.496.0578
www.developingchild.harvard.edu
www.developingchild.net

EARLY STEPS ELIGIBILITY DATA COLLECTION **June 1, 2010 thru June 30, 2010**

STATEWIDE EARLY STEPS

Chronologic Months of Age:	**0 - 11**	**12- 23**	**24 - 36**	**TOTAL**
NUMBER OF CHILDREN EVALUATED	376	550	747	1673
CURRENT ELIGIBILITY CRITERIA				
Number of Children Made Eligible due to Established Condition	136	40	28	204
Number of Children Made Eligible due to determination of Percent Delay	3	19	20	42
Number of Children Made Eligible based on Protocol Scores	94	346	460	900
Number of Children Made Eligible based on Informed Clinical Opinion	22	13	29	64
TOTAL ELIGIBLE	255	418	537	1210
Number of Children determined Not Eligible	121	132	210	463
Audit Total - Must equal the Number of Children Evaluated	376 0	550 0	747 0	1673 0
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010				
Number of Children Eligible due to Established Condition	140	42	29	211
Number of Children Eligible based on Protocol Scores	73	271	332	676
Number of Children Eligible based on Informed Clinical Opinion	22	16	43	81
TOTAL ELIGIBLE	235	329	404	968
Number of Children Not Eligible	141	221	343	705
Audit Total - Must equal the Number of Children Evaluated	376 0	550 0	747 0	1673 0

Percentage Reduction in Eligibility with New Eligibility Criteria

EARLY STEPS ELIGIBILITY DATA COLLECTION

STATEWIDE EARLY STEPS

Difference and Percentage Change

Chronologic Months of Age:	**0 - 11**	**12- 23**		**24 - 36**		**TO**
NUMBER OF CHILDREN EVALUATED						
CURRENT ELIGIBILITY CRITERIA						
Number of Children Made Eligible due to Established Condition						
Number of Children Made Eligible due to determination of Percent Delay						
Number of Children Made Eligible based on Protocol Scores						
Number of Children Made Eligible based on Informed Clinical Opinion						
TOTAL ELIGIBLE	67.82%		76.00%		71.89%	
Number of Children determined Not Eligible						
Audit Total - Must equal the Number of Children Evaluated						
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010						
Number of Children Eligible due to Established Condition		2		1		7
Number of Children Eligible based on Protocol Scores		-75		-128		-224
Number of Children Eligible based on Informed Clinical Opinion		3		14		17
TOTAL ELIGIBLE	62.50%	-89	59.82%	-133	54.08%	-242
Number of Children Not Eligible		89		133		242
Audit Total - Must equal the Number of Children Evaluated						
Percentage Reduction in Eligibility with New Eligibility Criteria	**-5.32%**	**-16.18%**		**-17.80%**		**-14.**

EARLY STEPS ELIGIBILITY DATA COLLECTION

STATEWIDE EARLY STEPS

		North Central			N
Chronologic Months of Age: TAL		**0 - 11**	**12- 23**	**24 - 36**	**0 - 11**
NUMBER OF CHILDREN EVALUATED		5	16	22	15
CURRENT ELIGIBILITY CRITERIA					
Number of Children Made Eligible due to Established Condition		2	1	0	5
Number of Children Made Eligible due to determination of Percent Delay		0	0	0	0
Number of Children Made Eligible based on Protocol Scores		0	9	15	7
Number of Children Made Eligible based on Informed Clinical Opinion		1	2	0	0
TOTAL ELIGIBLE	72.33%	3	12	15	12
Number of Children determined Not Eligible		2	4	7	3
Audit Total - Must equal the Number of Children Evaluated		5	16	22	15
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010					
Number of Children Eligible due to Established Condition		2	2	0	5
Number of Children Eligible based on Protocol Scores		0	7	13	6
Number of Children Eligible based on Informed Clinical Opinion		0	0	0	1
TOTAL ELIGIBLE	57.86%	2	9	13	12
Number of Children Not Eligible		3	7	9	3
Audit Total - Must equal the Number of Children Evaluated		5	16	22	15
Percentage Reduction in Eligibility with New Eligibility Criteria	**47%**	-20.00%	-18.75% -13.95%	-9.09%	0.00%

EARLY STEPS ELIGIBILITY DATA COLLECTION	lortheastern		Treasure Coast		
STATEWIDE EARLY STEPS					
Chronologic Months of Age:	**12- 23**	**24 - 36**	**0 - 11**	**12- 23**	**24 - 36**
NUMBER OF CHILDREN EVALUATED	32	57	43	62	92
CURRENT ELIGIBILITY CRITERIA					
Number of Children Made Eligible due to Established Condition	5	2	11	5	0
Number of Children Made Eligible due to determination of Percent Delay	0	0	2	7	4
Number of Children Made Eligible based on Protocol Scores	19	38	11	33	51
Number of Children Made Eligible based on Informed Clinical Opinion	0	1	0	0	1
TOTAL ELIGIBLE	24	41	24	45	56
Number of Children determined Not Eligible	8	16	19	17	36
Audit Total - Must equal the Number of Children Evaluated	32	57	43	62	92
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010					
Number of Children Eligible due to Established Condition	5	2	10	5	0
Number of Children Eligible based on Protocol Scores	18	32	7	19	26
Number of Children Eligible based on Informed Clinical Opinion	1	6	0	0	5
TOTAL ELIGIBLE	24	40	17	24	31
Number of Children Not Eligible	8	17	26	38	61
Audit Total - Must equal the Number of Children Evaluated	32	57	43	62	92
	0.00%	-1.75%	-16.3%	-33.9%	-27.2%
Percentage Reduction in Eligibility with New Eligibility Criteria	-0.96%			-26.9%	

EARLY STEPS ELIGIBILITY DATA COLLECTION	Western Panhandle			West Central		
STATEWIDE EARLY STEPS						
Chronologic Months of Age:	**0 - 11**	**12- 23**	**24 - 36**	**0 - 11**	**12- 23**	**24 - 36**
NUMBER OF CHILDREN EVALUATED	20	17	18	30	39	55
CURRENT ELIGIBILITY CRITERIA						
Number of Children Made Eligible due to Established Condition	11	1	0	18	0	0
Number of Children Made Eligible due to determination of Percent Delay	0	0	0	0	0	0
Number of Children Made Eligible based on Protocol Scores	3	10	11	9	27	34
Number of Children Made Eligible based on Informed Clinical Opinion	0	0	3	0	0	4
TOTAL ELIGIBLE	14	11	14	27	27	38
Number of Children determined Not Eligible	6	6	4	3	12	17
Audit Total - Must equal the Number of Children Evaluated	20	17	18	30	39	55
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010						
Number of Children Eligible due to Established Condition	11	1	0	18	0	0
Number of Children Eligible based on Protocol Scores	3	8	7	6	21	20
Number of Children Eligible based on Informed Clinical Opinion	0	0	1	1	1	1
TOTAL ELIGIBLE	14	9	8	25	22	21
Number of Children Not Eligible	6	8	10	5	17	34
Audit Total - Must equal the Number of Children Evaluated	20	17	18	30	39	55
Percentage Reduction in Eligibility with New Eligibility Criteria	0.00%	-11.76%	-33.33%	-6.67%	-12.82%	-30.91%
		-14.55%			-19.35%	

EARLY STEPS ELIGIBILITY DATA COLLECTION	Southwest Florida			Gulf Centra	
STATEWIDE EARLY STEPS					
Chronologic Months of Age:	**0 - 11**	**12- 23**	**24 - 36**	**0 - 11**	**12- 23**
NUMBER OF CHILDREN EVALUATED	12	33	29	32	36
CURRENT ELIGIBILITY CRITERIA					
Number of Children Made Eligible due to Established Condition	4	1	0	7	2
Number of Children Made Eligible due to determination of Percent Delay				1	3
Number of Children Made Eligible based on Protocol Scores	8	27	20	5	19
Number of Children Made Eligible based on Informed Clinical Opinion	0	1	0	5	1
TOTAL ELIGIBLE	12	29	20	18	25
Number of Children determined Not Eligible	0	4	9	14	11
Audit Total - Must equal the Number of Children Evaluated	12	33	29	32	36
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010					
Number of Children Eligible due to Established Condition	5	1	0	7	2
Number of Children Eligible based on Protocol Scores	4	20	15	5	18
Number of Children Eligible based on Informed Clinical Opinion	1	0	0	4	1
TOTAL ELIGIBLE	10	21	15	16	21
Number of Children Not Eligible	2	12	14	16	15
Audit Total - Must equal the Number of Children Evaluated	12	33	29	32	36
Percentage Reduction in Eligibility with New Eligibility Criteria	-16.7%	-24.2% -20.3%	-17.2%	-6.25%	-11.11% -9.17%

EARLY STEPS ELIGIBILITY DATA COLLECTION		Space Coast			
STATEWIDE EARLY STEPS					
Chronologic Months of Age:	**24 - 36**	**0 - 11**	**12- 23**	**24 - 36**	**0 - 11**
NUMBER OF CHILDREN EVALUATED	41	12	30	26	55
CURRENT ELIGIBILITY CRITERIA					
Number of Children Made Eligible due to Established Condition	4	3	0	1	13
Number of Children Made Eligible due to determination of Percent Delay	2	0	0	0	
Number of Children Made Eligible based on Protocol Scores	25	4	20	20	8
Number of Children Made Eligible based on Informed Clinical Opinion	1	2	2	1	7
TOTAL ELIGIBLE	32	9	22	22	28
Number of Children determined Not Eligible	9	3	8	4	27
Audit Total - Must equal the Number of Children Evaluated	41	12	30	26	55
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010					
Number of Children Eligible due to Established Condition	4	3	0	1	13
Number of Children Eligible based on Protocol Scores	22	2	9	10	8
Number of Children Eligible based on Informed Clinical Opinion	2	1	5	4	7
TOTAL ELIGIBLE	28	6	14	15	28
Number of Children Not Eligible	13	6	16	11	27
Audit Total - Must equal the Number of Children Evaluated	41	12	30	26	55
Percentage Reduction in Eligibility with New Eligibility Criteria	-9.76%	-25.00%	-26.67%	-26.92%	0.00%
			-26.47%		

EARLY STEPS ELIGIBILITY DATA COLLECTION	Gold Coast			Central Florida		
STATEWIDE EARLY STEPS						
Chronologic Months of Age:	**12- 23**	**24 - 36**		**0 - 11**	**12- 23**	**24 - 36**
NUMBER OF CHILDREN EVALUATED	40	86		28	34	38
CURRENT ELIGIBILITY CRITERIA						
Number of Children Made Eligible due to Established Condition	3	4		11	4	4
Number of Children Made Eligible due to determination of Percent Delay				0	0	0
Number of Children Made Eligible based on Protocol Scores	13	38		8	22	24
Number of Children Made Eligible based on Informed Clinical Opinion	2	7		3	4	6
TOTAL ELIGIBLE	18	49		22	30	34
Number of Children determined Not Eligible	22	37		6	4	4
Audit Total - Must equal the Number of Children Evaluated	40	86		28	34	38
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010						
Number of Children Eligible due to Established Condition	3	4		11	4	4
Number of Children Eligible based on Protocol Scores	10	27		5	18	15
Number of Children Eligible based on Informed Clinical Opinion	2	7		4	4	6
TOTAL ELIGIBLE	15	38		20	26	25
Number of Children Not Eligible	25	48		8	8	13
Audit Total - Must equal the Number of Children Evaluated	40	86		28	34	38
	-7.50%	-12.79%		-7.14%	-11.76%	-23.68%
Percentage Reduction in Eligibility with New Eligibility Criteria	-7.73%				-15.00%	

EARLY STEPS ELIGIBILITY DATA COLLECTION	Bay Area			Southernmost C	
STATEWIDE EARLY STEPS					
Chronologic Months of Age:	**0 - 11**	**12- 23**	**24 - 36**	**0 - 11**	**12- 23**
NUMBER OF CHILDREN EVALUATED	37	69	84	17	53
CURRENT ELIGIBILITY CRITERIA					
Number of Children Made Eligible due to Established Condition	17	11	5	6	1
Number of Children Made Eligible due to determination of Percent Delay	0	0	0	0	9
Number of Children Made Eligible based on Protocol Scores	10	40	49	4	39
Number of Children Made Eligible based on Informed Clinical Opinion	1	1	2	1	0
TOTAL ELIGIBLE	28	52	56	11	49
Number of Children determined Not Eligible	9	17	28	6	4
Audit Total - Must equal the Number of Children Evaluated	37	69	84	17	53
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010					
Number of Children Eligible due to Established Condition	18	12	6	6	1
Number of Children Eligible based on Protocol Scores	5	27	35	4	34
Number of Children Eligible based on Informed Clinical Opinion	0	1	2	1	1
TOTAL ELIGIBLE	23	40	43	11	36
Number of Children Not Eligible	14	29	41	6	17
Audit Total - Must equal the Number of Children Evaluated	37	69	84	17	53
Percentage Reduction in Eligibility with New Eligibility Criteria	-13.5%	-17.4%	-15.5%	0.0%	-24.5%
		-15.8%			-21.3%

EARLY STEPS ELIGIBILITY DATA COLLECTION	Coast	Big Bend				No
STATEWIDE EARLY STEPS						
Chronologic Months of Age:	**24 - 36**	**0 - 11**	**12- 23**	**24 - 36**		**0 - 11**
NUMBER OF CHILDREN EVALUATED	66	17	18	25		19
CURRENT ELIGIBILITY CRITERIA						
Number of Children Made Eligible due to Established Condition	1	8	1	1		8
Number of Children Made Eligible due to determination of Percent Delay	14	0	0	0		0
Number of Children Made Eligible based on Protocol Scores	40	3	15	17		5
Number of Children Made Eligible based on Informed Clinical Opinion	0	1	0	2		1
TOTAL ELIGIBLE	55	12	16	20		14
Number of Children determined Not Eligible	11	5	2	5		5
Audit Total - Must equal the Number of Children Evaluated	66	17	18	25		19
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010						
Number of Children Eligible due to Established Condition	1	8	1	1		8
Number of Children Eligible based on Protocol Scores	32	2	14	11		5
Number of Children Eligible based on Informed Clinical Opinion	6	1	0	2		1
TOTAL ELIGIBLE	39	11	15	14		14
Number of Children Not Eligible	27	6	3	11		5
Audit Total - Must equal the Number of Children Evaluated	66	17	18	25		19
Percentage Reduction in Eligibility with New Eligibility Criteria	-24.2%	-5.9%	-5.6%	-24.0%		0.00%
			-13.3%			

EARLY STEPS ELIGIBILITY DATA COLLECTION	orth Beaches		North Dade		
STATEWIDE EARLY STEPS					
Chronologic Months of Age:	**12- 23**	**24 - 36**	**0 - 11**	**12- 23**	**24 - 36**
NUMBER OF CHILDREN EVALUATED	26	30	34	45	78
CURRENT ELIGIBILITY CRITERIA					
Number of Children Made Eligible due to Established Condition	1	1	12	4	5
Number of Children Made Eligible due to determination of Percent Delay	0	0	0	0	0
Number of Children Made Eligible based on Protocol Scores	19	22	9	34	56
Number of Children Made Eligible based on Informed Clinical Opinion	0	0	0	0	1
TOTAL ELIGIBLE	20	23	21	38	62
Number of Children determined Not Eligible	6	7	13	7	16
Audit Total - Must equal the Number of Children Evaluated	26	30	34	45	78
ELIGIBILITY CRITERIA EFFECTIVE JULY 1, 2010					
Number of Children Eligible due to Established Condition	1	1	15	4	5
Number of Children Eligible based on Protocol Scores	17	18	11	31	49
Number of Children Eligible based on Informed Clinical Opinion	0	0	0	0	1
TOTAL ELIGIBLE	18	19	26	35	55
Number of Children Not Eligible	8	11	8	10	23
Audit Total - Must equal the Number of Children Evaluated	26	30	34	45	78
	-7.69%	-13.33%	14.71%	-6.67%	-8.97%
Percentage Reduction in Eligibility with New Eligibility Criteria	-8.00%			-3.18%	