Early Intervention Programs for Children and Families: Theoretical and Empirical Bases Supporting their Social and Economic Efficiency

Programas de Intervención Temprana para Niños y Familias: Bases Teóricas y Empíricas que Sustentan su Eficiencia Social y Económica

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Abstract. The prenatal period and the early years of life have an extraordinary importance on the physical and psychological well-being not only in the infancy, but throughout the life cycle. There is strong empirical evidence that early life is highly vulnerable to the negative effects of adverse experiences or toxic stress as maternal prenatal anxiety or child maltreatment. Research in the field of developmental neurobiology provides important keys about the mechanisms across these experiences affect the process of child development provoking alterations and dysfunctions in brain architecture. Such alterations tend to be persistent and increase the risk of physical, cognitive, social and emotional problems along infancy, adolescence and adulthood. The evidences clearly support the need and social relevancy of implementing early intervention preventive programs for children and families who are at risk for experiencing toxic stress. Such policies and programs should begin as early as possible in order to reduce or avoid the need of most costly and less effective remediation programs.
Keywords: early intervention, prenatal stress, toxic stress.

Resumen. El período prenatal y los primeros años de vida tienen una extraordinaria relevancia en la salud física y psicológica no sólo en la infancia, sino a lo largo del ciclo vital. Hay numerosas evidencias empíricas de que en este período el ser humano es altamente vulnerable a los efectos negativos de determinadas experiencias adversas (o lo que se denomina "estrés tóxico"), entre las que se pueden destacar la ansiedad materna prenatal o las situaciones de maltrato o negligencia en la temprana infancia. La investigación llevada a cabo desde la neurobiología evolutiva aporta claves importantes acerca de los mecanismos a través de los cuales dichas experiencias afectan el proceso del desarrollo infantil provocando alteraciones y disfunciones en la arquitectura cerebral. Dichas alteraciones tienden a ser persistentes e incrementan el riesgo de desórdenes y problemas físicos, cognitivos, sociales y emocionales a lo largo de la infancia, adolescencia y madurez. Las evidencias apuntan claramente la necesidad y relevancia social de desarrollar programas preventivos de intervención temprana con los niños y familias en situación de vulnerabilidad. Tales políticas y programas deben iniciarse lo antes posible para reducir o evitar la necesidad de desarrollar posteriormente intervenciones rehabilitadoras, que resultan más costosas y menos efectivas.
Palabras clave: estrés prenatal, estrés tóxico, intervención temprana.

Present and future welfare and progress in society depend on the healthy development of its members, meaning by health not only the absence of disease, but also availability of personal resources so that individuals are given the chance to adapt to the changes and challenges of everyday life, face adversity, possess a feeling of personal well-being, and participate and interact with their environments in an active and productive way (National Scientific Council on the Developing Child, 2010).

A comprehensive body of research supports the relevance of the prenatal period and the first five years of life for both physical and psychological health: what takes place in these stages provides the foundation for physical, cognitive and emotional development throughout life (National Scientific Council on the Developing Child, 2010; Shonkoff et al., 2012). However, the process of child development presents, as Shonkoff and Phillips (2000) stated, an unavoidable paradox: it is strong and vigorous, but at the same time

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highly vulnerable to the influence of adverse experiences which can have long-lasting negative effects on physical and psychological well-being. The present article shows some of the evidence from scientific research on the long-term impact of adverse experiences at the prenatal period and earliest years, with a special focus on the neurobiological mechanisms explaining how that influence takes place. The article also stresses the social relevance of implementing both prevention and early intervention services for vulnerable children and families, and reviews the outcomes of some of them.

The Effect of Adverse Experiences and Toxic Stress on Early Childhood Development

Adverse experiences may produce disturbances in the acquisition of the abilities and skills expected in the development process. When these experiences disrupt the successful completion of crucial developmental transitions and milestones, they do not only affect the particular abilities and skills specific to the developmental stage in which they occur, but they also increase the likelihood of subsequent difficulties and maladaptation in later stages. The organizational perspective on child development provides a useful framework to understand such process (Cicchetti, 1989; Cicchetti & Toth, 2005), although the relationship between early and later disturbances in human development and functioning is far from inevitable.

Research has identified some of the experiences that may disrupt significantly prenatal and early child development and produce a long-lasting negative impact on physical and psychological health. But not all adverse or stressful experiences have a negative impact on development. Exposure to them is frequent across the lifespan. Stress is an inevitable part of life, and the ability to cope with it is essential for survival as well as an important part of the developmental process.

In early childhood, the negative effect of stressful events depends on the intensity and duration of the physiological response to stress. The more intense and prolonged, the higher the probability of psychophysiological disorders associated to long-term and significant impairments in physical and psychological development (National Scientific Council on the Developing Child, 2005; Shonkoff et al., 2012). Many authors have adopted the stress-response taxonomy formulated by the National Scientific Council on the Developing Child in 2005, which differentiates between positive stress, tolerable stress and toxic stress. *Positive stress* refers to short-term, moderate, adverse situations (e.g., facing frustration, first day at school, meeting new people), which are frequently experienced by children and can become positive learning experiences if children have support from caring adults to cope with them satisfactorily and develop a sense of mastery. In these circumstances, physiological activation in response to the stressful event is brief and moderate in intensity, and support by adults helps the children to bring the activation of their stress-response systems down to baseline. These experiences give children valuable opportunities to observe, learn and practice healthy and adaptive responses to adverse experiences. *Tolerable stress* is also associated to a short-term exposure to adverse experiences, but with higher levels of adversity and threat for the physical and psychological health than in the previous type (e.g., conflictive separation or divorce, natural disaster, death or serious illness of a loved person). Also in these instances, disposition of caring and supportive adults becomes a key protective factor by helping children to cope with the adverse experience, develop a sense of mastery over it, and avoid excessive activation of their stress-response systems. In these cases, children frequently present *localized effects* (Finkelhor, 1995), that is, specific symptoms to adverse experiences that can be intense and persistent, but which do not interfere significantly with their development. The third and most dangerous type of stress response is the *toxic stress,* which occurs when the adverse experiences affecting the child are persistent, repetitive, uncontrolled and/or lacking the support of caring adults to cope with them, so that the child is unable to manage adequately and an intense and prolonged over-activation of the stress-response system occurs. In these instances, child development can be negatively affected in a deeper way, especially when there is an accumulation of stressors and interruption of important developmental transitions occurs (National Scientific Council on the Developing Child, 2005).

Research in the field of developmental neurobiology has provided valuable insights to better understand the mechanisms through which adverse experiences and toxic stress in prenatal and early years become a risk for long-term physical and psychological health. Apart from short-term changes in observable behaviour, there is strong evidence that toxic stress can produce less visible but stable changes in brain architecture and functioning which increase the risk of physical or psychological disorders, particularly when experienced during periods of rapid brain development (Gunnar & Quevedo, 2007; Mustard, 2006; National Scientific Council on the Developing Child, 2005, 2007, 2010; Pechtel & Pizzagalli, 2011; Shonkoff et al., 2012).

Brain growth and development starts at the prenatal period and, though it continues throughout childhood and adolescence, its main development takes place during the first years of life. This process involves creating, strengthening and discarding neural connections, and develops in a sequential and hierarchical manner, starting from the most simple brain regions

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and functions to the most complex ones. These different regions develop, organize and reach complete functionality at different times of development (sensitive periods), always in constant adaptation to the environment. The neural connections most frequently used are strengthened, while the potential connections not used or not properly stimulated do not activate or are discarded. Genetics provides a plan for brain development, but environment has the power to alter it: brain architecture modifies to adapt to the specific needs and characteristics of the environment (McCain, Mustard, & McCuaig, 2011; Mustard, 2010; National Scientific Council on the Developing Child, 2007). These modifications have an adaptive function, although in some cases, such as toxic stress adaptation, they can become maladaptive (McCrory, De Brito, & Viding, 2010).

Research has shown that toxic stress has an important impact on brain structure and functioning by decreasing in some cases the brain size and, most importantly, by activating the secretion of stress hormones, mainly cortisol. It has been found that persistent high levels of cortisol disrupt the neural growth and the formation of synapses, and causes modifications in the limbic system (mainly the hippocampus, the prefrontal cortex and the amygdale). These circumstances are linked to language, cognitive and socio-emotional impairments in childhood, more difficulties in adaptation and higher reactivity to stressful events even when these are of medium intensity, and immune system problems expressed in infectious, inflammatory and autoimmune diseases (Gunnar, Herrera, & Hostinar, 2009; Gunnar & Quevedo, 2007; McCain et al., 2011; Pechtel & Pizzagalli, 2011; Shonkoff et al., 2012).

Toxic stress effects in early childhood may persist into adulthood, including increased risk of depressive disorders, anxiety, post-traumatic stress disorder and physical problems such as cardiovascular diseases, type II diabetes or hypertension (Mustard, 2006). There are many studies supporting this association, although most of them use a retrospective approach. Among these we find the Adverse Childhood Experiences (ACE) Study (Middlebrooks & Audage, 2008), carried out with 17,000 adults in order to explore the link between childhood stressors and adult health. The adverse experiences analysed included child maltreatment, exposure to intimate partner violence, household substance abuse, household mental illness, and parental separation, divorce or imprisonment. Apart from a high frequency of these adverse experiences in childhood (according to the reports by the adult participants) the study found a strong association between these and a wide range of difficulties in later life, such as physical disorders (e.g., cardiovascular, breathing, and sexually transmitted diseases), illicit drug use, alcoholism, depression, partner violence, sexual promiscuity, unwanted pregnancies and suicide attempts. The study also found a positive relationship between the number of adverse experiences suffered and the risk of health problems at a later stage. This finding has also been confirmed in several longitudinal studies (Caspi, Harrington, Moffitt, Milne, & Poulton, 2006; Horwitz, Widom, McLaughlin, & White, 2001; Schilling, Aseltine, & Gore, 2007), although some analyses lead to think that the relevant factor could be not as much the quantity or accumulation of adverse experiences, but their severity or intensity (Schilling, Aseltine, & Gore, 2008).

Child maltreatment is one of the most severe adverse experiences a child may suffer, especially when occurs during sensitive stages of development. Moreover, it is frequent the concurrence of various types of maltreatment -physical, psychological and/or sexual- (Gilbert et al., 2009; Higgins & McCabe, 2001), other types of victimization, and associated adverse circumstances such parental mental health problems or substance abuse, chaotic, unstable and disorganized family environments, or family violence (Finkelhor, Ormrod, & Turner, 2007).

In recent years, many studies on the neurobiological consequences of child maltreatment and its relationship with a wide range of physical, cognitive, social and emotional problems in childhood, adolescence and adulthood have been carried out. In some cases, the maltreatment involves a severe lack of cognitive and sensory stimulation. Such experiences may alter the brain capacity to use serotonin –associated to feelings of well-being and emotional stability-, decrease brain volume and growth, and produce a loss of neurons, all these circumstances being related to impaired cognitive, emotional, behavioural and social functioning. Research has also shown that repeated maltreatment associated to a constant activation of the neurobiological stress-response systems disrupts the developing brain circuits. The chronic stimulation of the brain response associated to fearfulness seems to cause this type of responses to remain fixed, so that the child victim of severe and chronic maltreatment is in a state of constant alertness and over-activation which prevents him/her from adapting to non-threatening environments, from acquiring the calmness needed to learn, and from getting involved in the activities needed to develop complex cognitive, behavioural and emotional functions, such as emotional regulation and impulse control. Furthermore, as mentioned previously, permanent activation of the stress-response system is related to high levels of stress hormones –glucocorticoids, and particularly cortisol-, which weaken the immune system, thus rendering children more vulnerable to infections and chronic health problems (Child Welfare Information Gateway, 2009; De Bellis, 2005; Gunnar & Fisher, 2006; National Scientific Council on the Developing Child, 2005; Perry, 2002; Teicher et al., 2003; Twardosz & Lutzker, 2010; Watts-English, Fortson, Gibler, Hooper, & De Bellis, 2006).

The effects of child maltreatment on brain development may persist through adolescence and adulthood.

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A lower development of the brain cortex identified in adolescents who have experienced maltreatment during childhood has been related to increased impulsive behaviour, propensity to taking risks, and delays in academic and social skills. For adults with a history of severe maltreatment during childhood, abnormalities in brain growth, in the hippocampus, in the limbic system and in the connection between the cerebral hemispheres have been identified. These conditions have been linked to higher risk of memory impairments and psychopathological problems, such as depression, post-traumatic stress disorder and dissociative and attention-deficit disorders (Child Welfare Information Gateway, 2009).

The Vulnerability of the Prenatal Period

The assumption that child development shapes through a highly complex process of interplay between biological and environmental factors (Sameroff, 2000) explains the inclusion of prenatal stress in the list of adverse experiences which can affect development negatively, in particular when other risk factors are also present. Under the generic term of 'prenatal stress', different situations have been included, such as maternal anxiety and depression, relational difficulties, stress at work, and natural or man-made disasters (Glover, 2011).

Research carried out with primates has consistently found that maternal stress during pregnancy results in long-term negative effects on their offspring (Schneider, Roughton, Koehler, & Lubach, 1999; Schneider, Moore, Kraemer, Roberts, & DeJesus, 2002). These studies confirm that prenatal stress is associated to lower birth weight, lower levels of attention and motor maturity, slower learning, and impaired emotional regulation in offspring. The earliest stage of gestation seems to present a special vulnerability to these effects, although they have also been observed during mid-to-late gestation.

An important number of controlled studies with humans have shown a link between maternal prenatal stress and anxiety, and ultrasonographically observed foetal behaviour. Although few studies have proved the influence of maternal stress measured in early pregnancy (12-21 weeks) on near term foetal behaviour, there are many empirical data available supporting these effects in subsequent gestational stages (Van den Berg, Mulder, Mennes, & Glover, 2005).

Some studies have found a link between prenatal stress and the newborn health. Maternal self-reports on negative life events, stress at work and anxiety symptoms seem to be associated to premature childbirth and a smaller size of the newborn, both risk factors for cognitive and social developmental problems (Wadhwa, 2005; Wadhwa, Sandman, & Garite, 2001; Wadhwa, Sandman, Porto, Dunkel-Schetter, & Garite, 1993).

Several studies have also explored the relationship between prenatal stress and neurological and behavioural outcomes during the newborn period. These studies have found that newborns from mothers with high anxiety levels showed a higher activation of the brain right frontal lobe –which may be linked to negative affectivity from childhood to adulthood-, and spent more time in deep-sleep phases and less time in calm and active alert (Field et al., 1985). In general, those studies show that maternal prenatal anxiety is positively associated to child regulation disorders expressed by a lower performance in the Neonatal Behavior Assessment Scale (Rieger et al., 2004; Brouwers, Van Baar, & Pop, 2001) and other neurological tests (Lou et al., 1994).

Several prospective studies have found an association between prenatal stress and cognitive and socioemotional problems in early childhood. Infants of mothers with high levels of prenatal anxiety show less positive interactions with their mothers (Field et al., 1985), higher levels of negative affection and higher motor activity when presented new toys (Davis et al., 2004), higher irritability and difficulty (Van den Bergh, 1990; Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2003), poorer language skills (Laplante et al., 2004), and lower scores in the Bayley Mental Development Index (Huizink et al., 2003). Findings from a relevant longitudinal study (Van den Bergh et al., 2005) suggest that high levels of maternal state prenatal anxiety at 12-22 weeks of pregnancy are related to impulsive cognitive style and lower scores in intelligence tests when children are 14-15 years old. Moreover, it is important to point out the results from the longitudinal study by Van den Bergh & Marcoen (2004), which found that prenatal anxiety during pregnancy explained 22% of the variance in symptoms of attention-deficit/hyperactivity disorder (ADHD) in 8-9 year-old children. Other studies have also found a link between maternal prenatal anxiety and ADHD, and more difficulties in attention regulation (O'Connor, Heron, Golding, Beveridge, & Glover, 2002; Huizink et al., 2003).

Findings from these studies could be explained by factors such as the transmission of genetic susceptibility to anxiety, the effect of maternal postpartum anxiety, the effect of pre or postnatal anxiety on maternal perception of child behaviour, or the influence of other associated variables, such as a higher use of tobacco or alcohol during pregnancy or a higher presence of neglectful behaviours or physical aggressions against the infant which could have affected negatively the process of brain development (Van den Bergh, 1990). However, it is important to point out that, although such factors are relevant to explain the difficulties observed in postnatal functioning, research suggests that prenatal stress has also a significant effect (Glover, 2011). For example, findings obtained by Van den Bergh et al. (2005), controlling the effect of postnatal

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anxiety, postnatal depression and postnatal stress, show a strong association between maternal prenatal anxiety and regulation disorders in the child. These findings support the hypothesis that *fetal programming* by maternal stress or anxiety is occurring in humans and that the effects of the prenatal environment interact with genetic factors to determine the phenotype at birth.

The *fetal programming* hypothesis (Barker, 2002; Barker, Forsen, Eriksson, & Osmond, 2002) states that uterine environment may disrupt the development of the foetus during particularly sensitive periods. According to this hypothesis, quality of intra-uterine life (1) might program a certain degree of susceptibility in the individual to develop illnesses at later periods, and (2) might be as relevant as the genetic pool when predicting future physical and mental performance. According to this approach, throughout the foetal development there are critical periods of vulnerability to adverse or unfavourable conditions. These vulnerable periods take place at different times for different tissues and organs, with the most-rapid division cells being the ones at the greatest risk.

The fetal programming hypothesis also includes several key ideas. Firstly, the effects of foetal conditions are persistent. Secondly, the effects on health remain latent for many years. Thirdly, fetal programming occurs by the influence of the environment on the epigenome, a biological process still not fully understood.

The epigenome can be described as a series of switches that allow (or not) certain parts of the genome to express. The intra-uterine period may be particularly important to set or adjust those switches (Petronis, 2010). The fetal programming hypothesis states that epigenetic foetal changes may have an adaptive goal. In an adverse environment, the developing organism tries to compensate for and adapt to the deficiencies in order to increase chances of survival (Gluckman & Hanson, 2005). However, disorders can appear when postnatal conditions become different from those the foetus got prepared for. Thus, for instance, inadequate nutrition in utero *programs* the foetus to have metabolic characteristics that can lead to future disease; individuals starved in utero, who adapt to this adverse circumstance, are more prone in adulthood to be overweight and suffer cardiovascular disease and diabetes (Barker, 2002).

Children from mothers with high levels of anxiety or stress at pregnancy would have alterations in the functioning of the hypothalamic-pituitary-adrenal (HPA) axis to make them more vigilant and aware of potential environmental threats after birth. However, this hypervigilance and shift in attention may become maladaptive (Talge, Neal, & Glover, 2007). Alterations in the HPA axis would tend to remain for a long period of time, as suggested by the results obtained from studies which have found, for example, an association between maternal prenatal anxiety and children's morning concentrations of cortisol at ten years of age (O'Connor et al., 2005) or the cortisol levels in response to moderate stressors in six-month-old babies (Huot, Brennan, Stowe, Plotsky, & Walker, 2004). The most widely accepted hypothesis suggests that abnormally high levels of maternal stress hormones, particularly glucocorticoids, can cross the placenta and affect foetal brain and nervous system development (Gitau, Cameron, Fisk, & Glover, 1998; Van den Bergh et al., 2005).

Results from several studies suggest that different gestational ages may present different levels of vulnerability to specific types of disorders. Thus, for example, an association between a greater vulnerability to schizophrenic disorders and extreme stress in the first three months of pregnancy has been found, as well as between ADHD and prenatal stress in later stages of pregnancy (Glover, 2011). Other studies have found greater vulnerability to prenatal stress in the tenth week for ADHD symptoms at the age of seven (Rodriguez & Bohlin, 2005), in the first three months for negative emotionality at the age of five (Martin, Noyes, Wisenbaker, & Huttunen, 1999), in the first six months for intellectual disorders at age two (Laplante et al., 2004), in the first six months for disorders at ages 8-9 and cognitive functioning at ages 14-15 (Van den Bergh & Marcoen, 2004), etc. Variability of results suggest that different mechanisms are operating at different stages of gestation, or else, this might be a consequence of methodological differences between studies (Van den Bergh et al., 2005).

It is not clear if maternal prenatal anxiety and stress affect boys and girls in different ways. Most studies do not deal with this issue. Findings from some studies suggest a greater vulnerability in boys (O'Connor et al., 2002). Nevertheless, results from the study by O'Connor, Heron, Golding, & Glover (2003) suggest a possible interaction between gender and development: while the effects of prenatal anxiety in ADHD symptoms at the age of four were observed only in boys, differences between genders disappeared when the same children were evaluated at the age of seven.

Reversibility of Neurological Effects of Pre- and Post-natal Toxic Stress

Negative effects of toxic stress on brain architecture are not always irreversible. Some reviews suggest that certain interventions –for example, providing economic and emotional support to children, behavioural and pharmacological therapies at older ages- can improve the HPA axis regulation, with subsequent improvements in behavioural and emotional adjustment (Gunnar et al., 2009). However, other studies suggest that the effects of extremely intense adverse experiences, in particular long and severe cognitive and emo-

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tional deprivation during early childhood, may be irreversible (Mustard, 2006).

Animal studies have shown that some epigenetic modifications can be remediated (Barros et al., 2004; Champagne & Curley, 2009; Maccari et al., 1995; Smythe, McCormick, & Meaney, 1996; Szyf, 2009). These studies suggest that the adverse effects of prenatal stress exposure could be buffered with a postnatal nurturing environment: animals with an optimal parental care show a reduction of the behavioural effects of stress (Meaney, 2001; O'Donnel, Larocque, Seckl, & Meaney, 1994). Beneficial effects of positive postnatal experiences -either naturally occurring or by experimental manipulation- are as powerful as the negative experiences effects; positive postnatal care and prenatal stress appear to have opposite effects on the HPA axis (Vallee et al., 1999).

Findings of studies conducted with humans suggest that negative effects of prenatal stress on foetal brain development might be moderated by the effect of attachment. Results from a recent longitudinal study carried out with 125 mother-child dyads (Bergman, Sarkar, Glover, & O'Connor, 2010; Bergman, Sarkar, & O'Connor, 2008) showed an inverse association between prenatal cortisol levels, indexed by amniotic fluid levels measured at week 17th of pregnancy, and standard scores from the Bayley cognitive development scale at 17 months of age. However, a moderating role of infant-parent attachment quality on the association between prenatal cortisol exposure and cognitive development was observed. Whereas prenatal cortisol exposure strongly predicted cognitive development in children with an insecure attachment history, the relationship between both variables was essentially zero among children with a secure attachment history. Furthermore, this interaction -and, therefore, the moderating effect of attachment- was independent of obstetric and psychosocial covariates, like stressful events.

In the same study, infant temperament was assessed through observational methodology at laboratory measuring intensity of child's fear reaction at an unpredicted situation. Results showed that correlation between antenatal stress and observed fearfulness varied according to attachment classification: a significant interaction for attachment and antenatal stress was observed indicating that the prediction from antenatal stress to fearfulness was significantly greater in the insecure-ambivalent group compared the other three attachment classifications. Findings suggest that insecure-ambivalent attachment enhances the association between exposure to prenatal stress and infant fearfulness intensity assessed at 17 months of age.

Results from this longitudinal study provides direct human evidence that prenatal maternal cortisol exposure -marker of the maternal anxiety level during pregnancy- predicts infant cognitive and emotional development and that this effect can be moderated and even completely eliminated by a positive childrearing (secure attachment bonding). It also provides relevant evidence that negative early caregiving -insecure attachment- accentuates the negative effect of antenatal stress. Taking together, findings suggest that early postnatal interventions may confer benefits to the child and that some prenatal effects may be modifiable by infant-parent attachment in the postnatal period.

The Need for a Paradigm Shift: from Rehabilitation to Early Intervention

Integrating previous information, following conclusions can be proposed:

1. Alterations in brain architecture and functioning due to adverse pre and postnatal environments appear to remain stable. From an organizational approach to brain and human development, time is a relevant variable affecting intervention prognosis.
2. Early childhood intervention programs designed to mitigate the factors that place children at risk of poor outcomes, should be considered as the most effective and maybe the unique strategy to avoid persistent negative effects of early adverse experiences.
3. Early intervention programs aimed to reduce the number or adversity of adverse experiences threatening the well-being of young children, and to promote adequate nurturing for them (e.g., secure attachment with supportive adults), can achieve positive outcomes in children's emotional and cognitive development.

A shift in thinking in the public health and social services sector, from a treatment paradigm to a prevention paradigm, is needed. A large part of the social and health public services are meant to provide services to children and their families when moderate to severe problems are identified in family relationships (for example, child maltreatment), child cognitive development, substance abuse, criminal behaviour, etc.

Scientific research provides reasons supporting effectiveness of programs carefully designed to improve early parental care and trigger significant effects on a wide range of aspects of child development (for a more comprehensive review see Olds, Sadler, & Kitzman, 2007). Evaluation of some early prevention programs has showed positive short, medium and late post-treatment outcomes.

Early intervention programs are usually home visiting programs starting at pregnancy or early after child birth. Probably, one of the most widely implemented programs in the US is *Healthy Start.* To be more precise, this is in fact a group of programs which follow the original program conducted in Hawaii (Duggan et al., 1999), which has been adapted in the different places where it has been implemented (Bugental et al.,

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2002; Duggan et al., 2004; Landsverk et al., 2002; Mitchell-Herzfeld, Izzo, Greene, Lee, & Lowenfels, 2005). The main goal of *Healthy Start* is prevention of child maltreatment. Results have been very diverse and not always as positive as expected. A review conducted by the Washington State Institute for Public Policy (Aos, Lieb, Mayfield, Miller, & Pennucci, 2004) concluded that *Healthy Start* is not able to achieve expected outcomes and that costs are higher than benefits. Results are disappointing, in part, because of high rates of participant attrition.

There are many other programs designed to avoid the negative consequences of child exposure -before or after birth- to adverse situations: *Parents as Teachers* (Parents as Teachers National Center, 2005), *Early Head Start* (Love et al., 2002), *UCLA Family Development Project* (Heinicke et al., 1999; Heinicke, Fineman, Ponce, & Guthrie, 2001). However, for purposes of the present article, findings obtained from the evaluation of two programs will be presented: the *Comprehensive Child Development Program* and the *Nurse-Family Partnership Program*. The latter one is described in more detail in another article included in this special issue.

The implementation of the *Comprehensive Child Development Program* is an initiative by the American government to improve the life-chances of children born into low-income families. It is a program delivered by paraprofessionals home visitors and try to improve the family's economic self-sufficiency, children care, health and development. Randomized controlled trials of CCDP were conducted for more than 2,000 families receiving treatment and findings suggested that, although the dropout rate was acceptable, hardly any substantial differences were found in any developmental area between children who had received the treatment and those who had not (Goodson, Layzer, St. Pierre, Bernstein, & Lopez, 2000). CCDP is an example of a program receiving important public funding which had not achieved expected results at the time it was assessed. Probably, negative outcomes were partly due to the lack of a previous theoretical model or a standardized pattern for the intervention implementation, but it was mainly due to the lack of controlled pilot administrations which would have allowed a previous assessment of its efficacy (Gilliam, Ripple, Zigler, & Leiter, 2000; Olds et al., 2007). CCDP experience showed that not every prevention program can achieve expected outcomes, and that it is no possible to know program efficacy without a scientific evaluative approach.

The Nurse-Family Partnership Program (NFP; Olds, 2002, 2006) is a good example of an early prevention program for high-risk families with very positive results. NFP is a home visiting program delivered by nurses with specialized training who intervene with mothers and children providing support, education and information to prevent child maltreatment, to promote the child health, and to improve parental skills and life quality of the families. Intervention starts during pregnancy and lasts until the child is two years old. NFP program has three major goals: to improve the outcomes of pregnancy by helping women improve their prenatal health (health maintenance, nutrition and exercises, substance abuse and mental health); to improve the child's health and development by helping parents provide more sensitive and competent care of the child; and to improve parental life-course by helping parents plan future pregnancies, complete their educations, and find work. Reports from randomized control trials conducted to assess short and long-term outcomes (including results from 15 to 19-year follow-ups) have shown consistent effects on prenatal health behaviors, parental care of the child, child maltreatment, child health and development, maternal life-course, and criminal involvement of the mothers and children. Positive outcomes have also been observed in mothers' income, domestic violence and maternal depressive symptoms.

Social and Economic Implications

The *Human Capital Theory* could be a useful framework to explain some of the conclusions arising from available data on the effects of the early childhood quality on the development over a person's life (Kilburn & Karoly, 2008). *Human capital* is defined as "the set of activities influencing the economic and psychological gains obtained by increasing people's competence" (Becker, 1975, p. 9) and includes everything related to people's productive capacity like knowledge, health, experience, competences, etc. "Transformation of human capital over successive periods is known as a *human capital-production process*" (Kilburn & Karoly, 2008, p. 6).

There is an important body of rigorous research -some has been presented in this article, and some in other articles in this special issue- which shows that some prevention and early intervention programs carried out during pregnancy and the early years, are able to:

- produce long-lasting positive outcomes in development, avoid the effects that adverse experiences and toxic stress could have without the intervention and, therefore, improve individual's physical and psychological well-being, and
- generate considerable savings for society since it increases human capital thus enabling society to generate progress, and avoids high expenses in late-treatment programs that can only yield very limited results.

Nowadays a large number of expert groups linked to relevant scientific organizations (Heckman, 2006, 2008; Knudsen, Heckman, Cameron, & Shonkoff, 2006; National Scientific Council on the Developing

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Child, 2004, 2007; RAND Corporation, 2005, 2008; Washington State Institute on Public Policy, 2011, 2012) is supporting the implementation of early childhood intervention policies. Empirical evidence from program evaluation studies conducted with the highest levels of methodological requirements can be helpful to support early childhood policies: early childhood intervention programs implemented to avoid the negative effects of pre- and perinatal adverse environments constitute a social investment improving individual well-being, societal well-being, and incomes of society.

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