This chapter reviews the literature examining fetal and infant neurobehavioral development. Basic fetal neurological development and neurobehavioral functioning are reviewed. Major fetal neurobehavioral milestones and their assessment are addressed and include fetal behavioral states, heart rate, movement, and responsivity to stimuli. The processes of neurological growth from birth to age 2 are reviewed. Infant neurobehavioral development is addressed and includes state regulation and sleep, physical growth and motor development, and the basic processes underlying social-emotional development. For fetus and infant, research examining the associations between neurobehavioral development and maternal distress and poverty is reviewed. The implications for future directions in fetal-infant neurobehavioral research are discussed.

Keywords: Prenatal development, fetus, infant, mother-infant, neurological development, physiology

This chapter on fetal and infant neurobehavioral development covers both periods with nearly equal weight, reflecting, in part, the greater accessibility of the fetus to research and, more significantly, the increasing emphasis on developmental roots for later functioning, including those that take hold during fetal development. For both the fetus and infant, we cover basic brain development and key neurobehavioral milestones, some of which overlap conceptually (i.e., state regulation), as well as micro- and macrolevel contextual factors that appear to influence the developmental trajectories at each time: maternal distress and poverty. While the organization covers first the fetus on all topics and then the infant, our aim is to emphasize the continuities between fetal and infant neurobehavior,
as well as their shared orientation and vulnerability to their external worlds.

Fetal Brain Development

Fetal brain development can be divided into three primary phases: embryonic, which begins at conception and extends through the eighth gestational week; the early fetal period, which extends approximately to midgestation; and the late fetal period lasting up until birth. By the end of the embryonic period, the basic structures of the brain and central nervous system (CNS) are established. The early fetal period is critical for the development of the neocortex because most cortical neurons are generated during this period, and many have migrated to their positions in the neocortex and begun to form essential connections for future neural circuits (Stiles & Jernigan, 2010; Tau & Peterson, 2010). The late fetal period sees significant specialization of each neural region and formation of different cortical layers, as well as overall maturation of the brain (Encha-Razavi & Sonigo, 2003; Tau & Peterson, 2010). At the midpoint of gestation, 20 weeks, the brain weighs approximately 20 g (as compared with 350–400 g at birth) and has all necessary structures for mature functioning, but has a smooth cortical plate because the gyri and sulci have just begun to form (Encha-Razavi & Sonigo, 2003; Fogliarini et al., 2005). These fissures begin to appear between 18 and 24 weeks and distinguish the four lobes of the brain. Development of secondary and tertiary sulci continues through the end of gestation. The corpus callosum, which begins to develop...
around the 12th week of gestation, is recognizable by 21 or 22 weeks and continues to develop for the remaining prenatal period (Encha-Razavi & Sonigo, 2003). Throughout this development, changes in gross morphology of the neural system are underpinned by changes occurring at the cellular level (Stiles & Jernigan, 2010).

During the first 2 weeks of postconception development, the embryo is a simple, oval-shaped, two-layered structure with two types of migrating cells, hypoblasts and epiblasts, forming different layers of the future fetus. The hypoblast is closest to the blastocyst cavity and made of cuboidal cells that will give rise to extraembryonic structures, including the lining of the yolk sac. The epiblast is adjacent to the trophoblast and gives rise to all three germ layers of the embryo: ectoderm, mesoderm, and endoderm. Some of the blastocyst cells do not migrate and instead remain in the epidermal layer, where they are transformed into what are termed neuromesenchymal stem cells, which will give rise to the brain and CNS. These stem cells also are referred to as the neural progenitor cells and, as a region, the neural plate. A complex cascade of molecular signaling engineers this and other differentiation and regional location of embryonic stem cells (Stiles & Jernigan, 2010). During the third week of gestation, between embryonic day 20–27, the first well-defined neural structure, the neural tube, is formed from the folding and fusion of the neural plate (Joseph, 2000; Tau & Peterson, 2010). The neural progenitor cells are located within the hollow cavity of the neural tube, which eventually will become the ventricle system of the brain. As such, this area is called the ventricular zone (VZ), which will be relevant for understanding future neuronal production. Importantly, several lifestyle factors, such as inadequate folic acid and vitamin B and the use of antiepileptic medications such as valproic acid (also used to treat psychiatric disorders) can cause neural tube defects leading to either spina bifida or anencephaly (Artama, Ribvnen, Gissler, Isojarvi, & Auvinen, 2006). Because the neural tube forms just a month postconception and is essential for normal development, seemingly benign exposures can dramatically impact the child’s future before a woman even knows she is pregnant.

By 4 gestational weeks, the rostral portion of the neural tube forms three primary vesicles, the prosencephalon, mesencephalon, rhombencephalon, which are the embryonic precursors to the forebrain, midbrain, and hindbrain, respectively. By 7 gestational weeks, five secondary vesicles emerge (see Figure 1). Specifically, the prosencephalon differentiates into the telencephalon (from which will emerge the cerebral cortex and basal nuclei) and diencephalon (the future thalamus and hypothalamus), and the rhombencephalon differentiates into the metencephalon (which will give rise to the pons and cerebellum) and the myelencephalon (from which will emerge the medulla). The mesencephalon does not further divide (Stiles & Jernigan, 2010). Many of the cranial nerves also are formed during this period. The transformation of the overall structure of the embryo reflects specific changes at the cellular level, specifically in neural patterning throughout all regions of the nervous system. Changes in the patterning of the CNS begin in this period, are accelerated in the late fetal period, and extend for many postnatal years (Stiles & Jernigan, 2010). Embryonic neural patterning—the differentiation of distinct brain areas based on their cellular organization and neuronal connectivity—affects all brain regions; by the end of the embryonic period, primitive patterning of the sensorimotor regions within the neocortex is established, major compartments within the diencephalic and midbrain regions have differentiated, and the segmental organization of the hindbrain and spinal column have been laid down (Stiles & Jernigan, 2010).

During the early and late fetal periods (postembryonic development ending at gestational week 8), the gross morphology of the developing brain undergoes dramatic changes, including moving from a smooth, “lissencephalic” structure toward obtaining the characteristic mature “texture” of elevated ridges (gyri) and depressions (sulci). During these periods, brain development centers on the processes of neuron production,
migration, connections, and differentiation.

The human brain contains billions of neurons, most of which are produced by midgestation (Stiles & Jernigan, 2010). During the embryonic period, neural progenitor cells in the VZ divide by what is called a “symmetrical” mode of division, which produces two identical neural progenitor cells. By the fetal period, the mode of cell division shifts to “asymmetrical,” which produces one progenitor cell and one neuron. The new progenitor cell remains and continues to divide, whereas the postmitotic neuron leaves the VZ to take its place in the developing neocortex. This migration ultimately results in the formation of an orderly six-layered structure known as the laminar structure of the cerebral cortex. Migration occurs almost exclusively in an “inside-out” manner in which deeper cortical layers are formed before more superficial ones. For the postmitotic neurons, migration occurs via radial glial cells that serve as guides on the path of neurons to their final destination. Other neurons that emerge from the ventral telencephalon will migrate tangentially, that is, parallel to the developing cerebral cortex and thalamus, using a variety of guidance molecules produced in local regions along their route to direct their movement into the cortex. Neuronal migration peaks between gestational weeks 12 and 20, is largely complete by weeks 26–29, and is a focus of study with respect to prenatal exposures altering future neurobehavioral development (see below) (Stiles & Jernigan, 2010; Tau & Peterson, 2010), particularly as psychopathology now is conceptualized as brain disorders involving neural circuits (Insel & Wang, 2010).

As part of their migration, neurons extend axons and dendrites to appropriate synaptic partners. Central to this formation of early synaptic connections are “scaffolding cells” (functioning similarly to radial glial cells) and molecular gradients. There is continual refinement and modification inherent in the early construction of synaptic connections, and they are often transient placeholders to the more stable connections that characterize mature circuits. By week 5 of gestation, the earliest synaptic connections are formed (Tau & Peterson, 2010). This occurs with neurons located in the first recognizable cortical layer, known as the preplate. The neurons in the preplate serve as initial synaptic targets for neuronal projections from the developing thalamus and brainstem. Neurons within the preplate form a functionally active neural circuit, although it is quite primitive. Within the preplate exists the subplate; by weeks 18–22, it is thicker than the preplate and cortical plate, which forms out of the preplate. The subplate is rich with synapses and shows evidence of the laminar organization. Here, in the subplate, neurons receive preliminary afferent inputs from the visual and somatosensory thalamus, cholinergic afferents from the basal forebrain, and monoaminergic afferents from the brainstem. These neurons have the necessary molecular components for functional γ-aminobutyric acid (GABA)ergic transmission, which is excitatory during much of early development, serving to depolarize neuroblasts and cortical neurons, thereby increasing their action potential. Subplate neurons are capable of propagating neural signals across monoaminergic, cholinergic, and glutamatergic synapses. It is clear that the architecture of the major neurotransmitter systems is in place by early gestation, although much remains to be discovered about their role in early cortical development. Moreover, this emerging understanding of early brain circuitry indicates that fetal exposure to factors that influence signaling, metabolism, or other elements of neurotransmitter physiology—such as drugs of abuse or prescription medications—may impact the development of neural circuits and neurotransmitter systems (Tau & Peterson, 2010).

Within the cortical plate, synaptic density grows by about 4% per week, until the peak transfer of afferent synaptic connections from the preplate to the cortical plate between weeks 26 and 28. Dendritic arborization and synaptogenesis accelerate in the late fetal period to produce a thickening of the developing cortex. At week 34, approximately 40,000 new synapses are formed every second, a process that continues into early postnatal life. Meanwhile, the subplate disappears by the end of the fetal period (Tau & Peterson, 2010).

The different layers of cortex contain different types of neurons. Early on, neural progenitor cells can produce any neuron type, but as development proceeds, they lose this capacity in a process termed “fate restriction,” which is believed to be controlled by cell intrinsic signaling, although this process still is poorly understood (Stiles & Jernigan, 2010). As it develops, the cortical plate acquires the organizational features of the mature cortex: lamination first exists in the primary sensory and motor cortices by week 25 and a full complement of vertical lamina in the developing cortex by week 32, including afferents of all of the major neurotransmitter systems and a diversity of differentiated glia and neuronal cell types, such as excitatory glutamatergic spiny pyramidal neurons and GABAergic nonpyramidal interneurons (Tau & Peterson, 2010). Depending on the specific cortical region, cortical layer I is formed between weeks 24 and 34 and layers III–IV between weeks 32 and 34. Each layer contains a unique array of cell types, “the morphology and laminar location of which dictate the pattern of local and distant projections that each cell may send and receive” (Tau & Peterson, 2010; p. 150) and thus shaping the micro- and
macrocircuits throughout the brain that subserve human functioning.

Finally, two other developmental processes play key roles in fetal brain development: cell death (apoptosis) and myelination. Although the number of neurons in the human brain peaks at 28 weeks, as many as half of these neurons die as a result of naturally occurring cell death. This culling of neurons is a regulated process although the reasons for the overproduction of neurons are not well understood. The well-documented effects of fetal alcohol exposure have been linked to increased neuroapoptosis in the developing brain (Farber, Creeley, & Olney, 2010), although alcohol exposure also has been shown to impair the function of cell adhesion molecule L1, which is important in neuronal migration and differentiation (Tau & Peterson, 2010). Myelin, which enhances the speed and accuracy of the transmission of information encoded in action potentials that propagate along neurons, is detected first in subcortical regions then in cortical regions between 20 and 28 weeks. Myelination may be sensitive to perinatal hypoxia or ischemia, as well as to environmental toxins such as drugs of abuse, suboptimal nutrition, and the effects of preterm birth, contributing to risk for poor neurodevelopmental outcomes (Tau & Peterson, 2010).

**Fetal Behavioral States**

Intrauterine life is not static but continuously varying with respect to changes in fetal movement, heart rate, hemodynamics, metabolism, eye movements, and the stimuli and exposures that the fetus encounters (Arduini, Rizzo, & Romanini, 1995). The dramatic transformation of the fetal brain from the embryonic period to birth subserves an equally remarkable behavioral change that confers organization to the shifting in utero world—the obtainment of identifiable and coherent fetal behavioral states. For the purposes of fetal neurobehavioral assessment, the identification of fetal states has two functions similar to those in infant state evaluation: (1) reflecting developmentally expected neural maturation that can support discrete, organized states and the transition between them; and (2) providing the context for the evaluation of other aspects of fetal neurobehavioral development by indicating the level of fetal arousal at the time of assessment (DiPietro, 2005).

Behavioral states consist of the identification of physiological and behavioral variables that are stable over time and repetitive within individual subjects, and in similar forms, across all subjects studied (Arduini et al., 1995). Following a developmental neurology approach to infant states developed in the 1970s by Prechtl (Prechtl, 1974), and aided by the ability to visualize the fetus via the introduction of ultrasound methods, various researchers began to identify behavioral cycles in the fetus based on the clustering of fetal movement, heart rate, and eye movement (Arduini et al., 1995; Nijhuis, Prechtl, Martin, & Bots, 1982). There are four distinct fetal behavioral states that approximate newborn states (noted in parentheses): 1F (quiet sleep): quiescence, sometimes interrupted by brief gross body movements, eye movement is absent, heart rate is stable with a small oscillation bandwidth, no accelerations, except in combination with a startle; 2F (active REM sleep) identified by frequent and periodic gross body movements such as stretches and movements of the extremities, eye movement is present, heart rate has a wider oscillation bandwidth and frequent accelerations that coincide with movement; 3F (quiet waking) gross body movements are absent, eye movement is present, heart rate has a wider bandwidth than in 1F yet no accelerations; 4F (active waking) noted for its vigorous continual activity, including many trunk rotations, eye movement is present, heart rate is unstable with large and long-lasting accelerations, often merging into sustained tachycardia (Nijhuis, 1995). Characteristics of neural activity observed in infant sleep states have been seen in baboon fetuses monitored using electroencephalogram (EEG) tools surgically implanted (Grieve, Myers, & Stark, 1994).

Mature fetal state profiles develop in the late third trimester, typically at approximately 36 weeks or beyond. Once fully developed, state transitions can be observed during which near-simultaneous shifting in the three parameters occurs. Prior to this point of maturation, discrete states can be observed, but the correspondence among the three parameters is less frequent, and transition between states is less well-defined. There is significant variability in the proportion of time fetuses spend in specific states, although some trends are evident: at all gestation ages, active sleep, 2F, is the most commonly observed state, followed by quiet sleep, 1F. Active wakefulness, 4F, is relatively rare and begins to emerge near term. Time spent in quiet wakefulness, 3F, is very rare, even near term (DiPietro, 2005; Nijhuis, 1995). In studies with the near-term fetal baboon, states 2F and 1F also were the most frequent (Grieve et al., 1994). The emergence of mature states follows the establishment of different layers of the fetal brain cortex, as well as the surges of neuronal proliferation and synaptic connections. However, the specific mechanisms underlying the maturation of these states are only now under study, with some data indicating that the...
preoptic areas of the hypothalamus and the forebrain are involved (Mohns, Karlsson, & Blumberg, 2006).

In full-term fetuses, prolonged periods without coincidence of physiological and behavioral factors are believed to indicate disruption of centrally mediated control mechanisms (Groome, Swiber, Atterbury, Bertz, & Holland, 1997). Fetuses of diabetic women with well-controlled glucose levels show a delay in the expected emergence of organized fetal states (Visser, Mulder, Bekedam, van Ballegooie, & Prechtl, 1986), whereas similar results are seen in those with intrauterine growth retardation (IUGR; Arduini et al., 1995). In addition, compared to healthy controls, IUGR fetuses are found to have more disorganized state transition, specifically longer intervals between shifts in behavioral states and discordant heart rate during such state transitions (Arduini et al., 1995). Finally, in a study building on the individual variation in fetal state development, DiPietro, Costigan, and Pressman (2002) showed that indicators of proportionately higher biobehavioral concordance (fetal heart-rate/movement concordance) predicted better biobehavioral regulation in the 2-week-old neonate. In another study, late third-trimester fetuses and 2-week-old newborns spent nearly identical proportions of time in quiet and active sleep (Groome et al., 1997). Taken together, these findings indicate the validity of fetal state as an approach to characterizing fetal neurobehavioral development, as well as underscore the phenomenon that aspects of neurobehavioral development measured in infants begin in the fetus and are accessible to evaluation (DiPietro, 2005). Aspects of state regulation can index individual differences that are maintained through postnatal development, as well as indicate typical versus atypical development.

**Other Milestones in Fetal Neurobehavioral Development and Their Assessment**

To ascertain developmental milestones in the fetus, as well as individual differences in these capacities, investigators have focused on one or more aspects of functioning within three general domains: fetal heart rate, movement, and responsivity to stimulation (DiPietro, 2005). As indicated, fetal state also is used to index development, and it is sometimes controlled for in evaluations using one of the other domains.

**Fetal Heart Rate**

For infants, as well as for adults and children, measurement of resting heart rate, and, in particular, indices of its variability, are used to characterize not only physical health, but also psychological well-being (Porges, 1997; Porges, Doussard-Roosevelt, & Maiti, 1994; Porges & Furman, 2011; Sloan et al., 2001; Smith, Limon, Gallo, & Ngu, 1996). For example, in the context of psychological challenge, adults high on hostility as well as those with borderline personality disorder show decreases in heart rate variability compared to healthy controls (Austin, Riniolo, & Porges, 2007; Sloan et al., 2001), while higher levels of resting heart rate variability, largely controlled by vagal activation (Bernston et al., 1997), are associated with more overall adaptive emotion regulation (Porges, 1997; Porges et al., 1994; Sloan et al., 1994; Smith et al., 1996).

Over the course of gestation, fetal heart rate declines (DiPietro, Irizarry et al., 2004; Pillai & James, 1990) while variability increases (DiPietro, Caulfield et al., 2004; DiPietro, Hodgson, Costigan, & Hilton, 1996c; Van Leeuwen, Lange, Bettemann, Gronemeyer, & Hatzmann, 1999). Specifically, from midpregnancy to near term, average resting fetal heart rate decreases by approximately 6 beats per minute (bpm); in one study, this decrease was from 146.8 bpm to 140.9 bpm (DiPietro, Hodgson, Costigan, & Hilton, 1996c), whereas another reported average fetal heart rate at 40 weeks at 130 bpm (Pillai & James, 1990). Heart rate variability, frequently computed based on standard deviation (DiPietro, Hodgson, Costigan, & Hilton, 1996c), increases more dramatically: in one study of 31 fetuses observed monthly for 50-minute ultrasound sessions from 20–38 weeks, short-term variability based on standard deviation increased at a rate of 5% per week until 32 weeks, then at 1.1% thereafter (DiPietro, Hodgson, Costigan, & Hilton, 1996c). Although the human heart begins to beat at 3 weeks post conception, the developmental changes in heart rate and variability reflect an increase in parasympathetic innervation of the heart, as well as changes in neural development, particularly myelination of cortical and vagal processes (Dalton, Dawes, & Patrick, 1983; Dawes, Houghton, Redman, & Visser, 1982; Sachis, Armstrong, Becker, & Bryan, 1982). By midgestation, fetal heart rate can be reliably collected for prolonged periods using Doppler-based electronic fetal monitors applied to the maternal abdomen. At this point, the fetal heart is substantial in size relative to other potential sources of artifact, and signal loss due to movement is not as common as it is at earlier time points (DiPietro, 2005). In the assessment of fetal neurobehavior, heart rate acceleration or deceleration in relation to stimuli often is used as a response variable (Buss et al., 2009; DiPietro, Costigan, Nelson, Gurewitsch, & Laudenslager, 2008;
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Kisilevsky, Pang, & Hains, 2000; S. Kisilevsky, Hains, Jacquet, Granier-Deferre, & Lecanuet, 2004; Lecanuet, Granier-Deferre, & Busnel, 1988; 1989; Lecanuet, Granière-Deferre, Jacquet, & DeCasper, 2000; Lecanuet & Schaal, 1996; Monk et al., 2004; 2010) as is resting fetal heart rate variability (DiPietro, Hodgson, Costigan, & Hilton, 1996c; DiPietro, K.A., Hilton, & Pressman, 1999). (See the section “Exposures” for examples of these findings.) Importantly, signal loss for detecting fetal heart rate can occur during instances of fetal movement, which is not randomly distributed (DiPietro, 2005). More active fetuses can generate less reliable heart rate data. Average heart rate signal loss has been observed to be 6% at 20 weeks of gestation, and between 1% and 5% at 28 weeks and beyond (DiPietro, Hodgson, Costigan, & Hilton, 1996c).

Fetal Movement

In the newborn, movement can be evaluated based on tone, posture, reflexes, and overall activity level (DiPietro, 2005); in the fetus, the primary focus is on different quantifications of spontaneous movement. More specifically, for psychological and developmental research, fetal movement has been investigated as a precursor of individual variation in activity, a commonly studied construct in temperament research (DiPietro, 2005; DiPietro, Hodgson, Costigan, & Johnson, 1996a).

Even in the embryonic and early fetal periods, spontaneous movement is present (de Vries, Visser, & Prechtl, 1982). As gestation continues, more complex movement patterns emerge, such as swallowing, yawning, breathing, hiccupping, head rotation, and leg movements, which are in place by the first trimester (de Vries & Fong, 2006; Roodenburg, Wladimiroff, van Es, & Prechtl, 1991). Eye movement appears at 14 weeks, whereas other movements specific to the face are seen later term, such as eyes opening at 26 weeks, and, nearer to term, grimacing, non-nutritive sucking, and tongue protrusion (de Vries & Fong, 2006). General movements, the most common form of movement and present from the first half of gestation, also increase to term, with changes in the duration of the movement and in pauses between movements increasing (de Vries & Fong, 2006). Fetal movement shows a circadian rhythm, peaking late in the evening (Arduini et al., 1987), whereas fetal breathing peaks following the mother’s mealtimes (de Vries, Visser, & Prechtl, 1988). Fetal movement declines over the course of gestation; according to one study, after 20 weeks, fetuses move approximately every minute and are active between 10% and 20% of the time (DiPietro, 2005; DiPietro, Caulfield, et al., 2004). There is some continuity between late fetal movement activity and newborn levels within the first 2–4 weeks of pregnancy (Groome et al., 1999), although somewhat less consistent results show level of movement and associations with temperament parameters in early infancy (DiPietro, Bornstein et al., 2002). For example, movement level at 36 weeks was positively associated with observed activity level more than 1 year later for boys, although it was inversely associated for girls; whereas for both sexes, overall fetal movement was negatively associated with distress to limitations at the same age (DiPietro, Bornstein et al., 2002).

Fetal movement, and the lag-time between it and a heart rate incursion, also has been used to index fetal maturation and group differences in neurobehavioral development (DiPietro, Caulfield, et al., 2004; DiPietro, Hodgson, Costigan, & Hilton, 1996c; DiPietro, Hodgson, Costigan, & Johnson, 1996b; DiPietro et al., 2010; Timor-Tritsch, Dierker, Zador, Hertz, & Rosen, 1978). Specifically, from 20 weeks through term, fetal motor activity is increasingly associated or “coupled” with accelerations in fetal heart rate; the strength of this “coupling” relationship is an indicator of fetal well-being, and reflects integration between somatic and cardiac functioning, as well as maturation of the autonomic nervous system (DiPietro, Hodgson, et al., 1996b). In several studies, DiPietro et al. have demonstrated an increase in coupling over gestation (DiPietro, Hodgson, Costigan, & Hilton, 1996c), as well as a positive association between fetal movement and heart rate coupling and newborn brainstem auditory evoked potentials (DiPietro et al., 2010). Lower levels of coupling, and in its rate of increase over gestation, have been identified in fetuses of lower socioeconomic status (SES; Pressman, DiPietro, Costigan, Shupe, & Johnson, 1998) and in those in Lima, Peru, versus Baltimore, Maryland, despite comparable prenatal care across both groups (DiPietro, Caulfield, et al., 2004). Hence, coupling may be an important predictor of subsequent infant development that is worthy of future research.

Pregnant women do not typically feel fetal movement until the 16th or 18th gestational week; with respect to assessment of fetal movements, women do not accurately judge their presence or absence (de Vries & Fong, 2006; Kisilevsky, Killen, Muir, & Low, 1991). Moreover, although doppler actigraphy provides reliable detection of fetal movement data, divergences in the quantification of movement bouts can make difficult direct comparisons of...
fetal movement data across labs (de Vries & Fong, 2006; DiPietro, 2005). There is another complication with respect to fetal neurobehavioral assessment based on movement and coupling criteria: incidences of fetal movement at the extreme ends of the range limit and increase the opportunities for associations with heart rate incursions, which could affect the coupling value (DiPietro, personal communication). And fetal movement can be associated with loss of the heart rate signal, which can bias data collection in very active fetuses.

Responsivity to Stimuli

Fetal response to stimuli can be elicited and tested directly and provides a window into fetal neurobehavioral functioning. In these stimulus-eliciting paradigms, the source of the stimuli varies (vibroacoustic, musical notes, maternal state changes), as does the response variable (heart rate change, movement, habituation). By 22 weeks, some fetuses show arousal to external stimuli, and by 30 weeks such responses can be reliably elicited (Leader, 1995). The demonstration of an elicited fetal response is intriguing with respect to investigations of the development of sensory capacities and emerging brain circuitries supporting information processing. However, there is some debate as to the interpretation of fetal responses, with one camp arguing caution (DeCasper, Granier-Deferrre, Fifer, & Moon, 2011), on the belief that the fetuses' seemingly complex behaviors are primarily reflective of brainstem reflexes and only minimal forebrain contribution (Joseph, 2000), and the other camp referring to fetuses as showing attention and discrimination and evoking the involvement of higher order brain processes (Kisilevsky & Hains, 2011). In what follows, we discuss fetal responses to standardized vibroacoustic and auditory probes, as well as to acute changes in maternal psychological state (laboratory-elicited stress and induced relaxation). As will be considered in the next section, more than the other milestones, the capacity for elicited responses (and subsequent regulation) underscores the fact that the roots of individual differences in neurobehavioral functioning—often independent of maternal or fetal pathology—emerge and are readily assessed prior to birth.

Vibroacoustic Stimuli: Fetal Movement, Heart Rate, and Habituation

A vibroacoustic stimulus, as opposed to an airborne sound stimulus (see below), is delivered on the maternal abdomen and includes both an airborne sound and a mechanical tactile vibration. When the vibroacoustic stimulus is intense (i.e., >85 decibel [dB]/sound pressure level [SPL]), a variety of fetal responses are elicited: startle, heart rate increase, abrupt state changes, decreased fetal breathing movements, and micturition (Visser & Mulder, 1993; Zimmer & Divon, 1993). Relative to intense stimulation, milder vibroacoustic sound stimuli generate less robust behavioral responses and have been used more consistently to chart developmental timing of the fetal response. For example, in a study of 60 healthy pregnant women carrying singleton fetuses using a vibrator with 64 dB SPL airborne sound, Kisilevsky, Muir, and Low (1992) showed that the response to this kind of stimulation begins at about 26 weeks of gestation, with movement activity coming online first, then a reliable heart rate acceleration at about 29 weeks; overall, the response pattern increases over a 6-week period, becoming fully mature at about 32 weeks. This vibroacoustic test, which involves sensory, motor, and/or heart rate responses, reflects higher level CNS activity than the observation of basal movement and heart rate functioning. Anencephalic fetuses may have normal movement and heart rate activity, yet they do not show a response to vibroacoustic stimulation (Leader, Baillie, Martin, & Vermeulen, 1982; Ohel, Simon, Linder, & Mor-Yosef, 1986).

Vibroacoustic stimulation also has been used to demonstrate habituation (learning, or the cessation of a behavioral response that occurs when an initially novel stimulus is presented repeatedly) in the human fetus and dishabituation (memory, or the recovery of a response to the original stimulus following the presentation of a second novel stimulus). Several studies indicate that fetuses habituate to external stimulation by approximately 25 weeks of gestation (Leader et al., 1982; Leader & Baillie, 1988; Leader, Stevens, & Lumbers, 1988; Madison, Madison, & Adubato, 1986). Habituation is associated with other indicators of the level of behavioral development independent of gestational age (Morokuma et al., 2004). Fetuses with Down syndrome take longer to habituate, and different patterns of habituation are associated with different degrees of Down syndrome and atypical neurodevelopment (Hepper & Shahidullah, 1992). One report, one of the few to include dishabituation and thus helping to distinguish true habituation from response fatigue (DiPietro, 2005), showed by changes in fetal heart rate that by 32 weeks fetuses were capable of habituating to a 63 dB stimulus and then dishabituating to it by showing a change in response to another novel stimulus delivered at 68 dB (Sandman, Wadhwa, Hetrick, Porto, & Peeke, 1997). However, the methodology has been subject to criticism, given the reliance on small changes in fetal heart
rate, as well as on the use of repeated stimulation (i.e., 40 times over 45 minutes; DiPietro, 2005).

Music and Voice Stimuli: Fetal Heart Rate

By 35 weeks, cochlear biomechanics and frequency selectivity are mature, enabling the fetus to process auditory stimuli (Eldredge & Salamy, 1996) and discriminate intensity, frequency, and spectra as the fetus nears term (Lecanuet et al., 2000). In a series of carefully executed studies, Lecanuet and his colleagues (Lecanuet & Schaaf, 1996; Lecanuet, Granier-Defere, Jacquet & Busnel, 1992) showed that fetuses between 36 and 40 weeks of gestation differentiated between male and female voices reading the same text based on their heart rate deceleration (an orienting response) to the change in speaker (Lecanuet et al., 1992). Other data indicate that fetuses as young as 28 weeks respond to music within 30 seconds of exposure to it, as evidenced by a heart rate increase in response to Brahams’s “Lullaby” delivered through a loudspeaker positioned near the maternal abdomen. However, by later in pregnancy, the fetal heart rate response shifted to a heart rate deceleration to the same stimulus, at least at lower decibel intensities (Kislevsky et al., 2004). Such findings are intriguing, but, as DeCasper et al. (2011) recently pointed out, teasing out the effects of external stimuli on fetal heart rate is extremely challenging, and thus characterizing responses as “orienting” and implying “paying attention” should be rendered cautiously; the science of fetal auditory processing is still in its infancy. On the other hand, a recent study exploited the continuity between fetal and infant development and the ease of newborn versus fetal assessment to demonstrate support for fetal auditory processing and memory development. Specifically, Granier-Defere, Bassereau, Ribeiro, Jacquet, and Decasper (2011) exposed 25 fetuses to a descending piano melody twice daily at 35, 36, and 37 weeks of gestation. Six weeks postpartum, the infants were exposed to the same music during quiet sleep, as were 25 control infants naïve to the piano music. Both groups of infants also heard a new stimulus, an ascending piano tune. Although all infants displayed a significant heart rate change, the infants exposed to the music as fetuses showed a cardiac deceleration that was two times larger than the deceleration elicited by the control music and the control responses to either piano melody (Granier-Defere et al., 2011). Other studies, on the basis of infant sucking responses instead of heart rate, have shown that newborns prefer their mothers’ voice over another woman’s (DeCasper & Fifer, 1980) and that 2-day-olds can differentiate the language they heard in utero compared to a novel one (Byers-Heinlein, Burns, & Werker, 2010).

Acute Changes in Maternal State: Fetal Heart Rate, Heart Rate Variability, and Movement

Fetal habituation and responses to music underscore the potential for environmental shaping of neurobehavioral development before birth. An age-old question is whether maternal psychological experiences similarly reach the fetus and if they, over the course of pregnancy, influence children’s developmental trajectories. One approach to this question is the acute induction of a specific maternal psychological “state,” such as stress or relaxation, and the identification of a concurrent fetal response. For example, in a study from 1967, maternal and fetal heart rate increases were induced by a sham protocol in which a subject was “told that she was ... breathing a gas which contained only half the amount of oxygen necessary to support fetal life, but that her normal body mechanisms would probably compensate for this altered environmental condition” (Copher & Huber, 1967). The authors concluded from their significant results based solely on eliciting anxiety from pregnant women that there is a “basis for concern regarding the emotional status of the subject when testing the effects of maternal [environmental] stimuli [i.e., actual changes in air oxygen levels] on the maternal–fetal relationship” (Copher & Huber, 1967). More recently, a few other laboratories have aimed to study the acute “transmission” of maternal experience to the fetus.

Two groups have used a common cognitive challenge assessing interference in reaction time, the Stroop color-word matching task, in a laboratory setting to induce acute stress in pregnant women while they and their fetuses are physiologically monitored (DiPietro, Costigan, & Guriewitsch, 2003; Monk et al., 2000; 2004; 2010). In results from DiPietro et al. (2003) with fetuses at 24 and 36 weeks’ gestation, the Stroop task led to significant increases in maternal heart rate and skin conductance (indicative of sympathetic activation), as well as to increases in fetal heart rate variability and a suppression of motor activity (but not to change in fetal heart rate). The maternal results declined from 24 to 36 weeks’ gestation while the magnitude of the fetal responses increased from the first to the second testing period. Because there was only one modest association between a maternal physiological variable (skin conductance) and fetal motor activity (a positive association), DiPietro et al. hypothesized a “sensory” versus “physiological” pathway by which maternal experience may impact the fetus. In the latter model, the primary mechanism would be direct physiological mediation. Changes in maternal cardiovascular activity would
contribute to changes in fetal heart rate or movement as a consequence of physiological processes aimed at maintaining homeostasis and/or reactivity indicative of a disruption in the typical physiological milieu. In contrast, in the sensory model, maternal physiological reactivity the fetus responds to the sensory stimulation of the maternal stress response i.e., the auditory stimuli from maternal heart rate and blood pressure changes. Data from other studies are, for the most part, consistent with DiPietro et al.’s results and lend support to the sensory interpretation.

In a series of studies also using the Stroop color-word matching task with pregnant women in the third trimester, Monk and her colleagues (Monk et al., 2000; 2004; 2010) found no change in fetal heart rate in response to women’s exposure to the cognitive challenge (in fetuses of psychiatrically healthy women; see below) and only minimal associations between maternal physiological reactivity and fetal responses (in these results, a modest association between changes in systolic blood pressure and fetal heart rate). Fetal movement and heart rate variability were not assessed. Monk et al. (2010) also examined an additional task for the participants, standardized changes in breathing rate, and found that stimulus to produce dramatic increases in fetal heart rate.

There were no direct associations between maternal and fetal physiology, yet robust changes in fetal heart rate occurred when women were alternatively breathing half as fast as typical or two times faster. Moreover, such changes in breathing, accompanied by abdominal and chest movement, may provide auditory as well as vestibular stimuli to the fetus. Taken together, these results are consistent with the hypothesis that changes in pregnant women’s cardiorespiratory activity (some of which may be associated with psychological state) function as stimuli, which the fetus perceives and to which it responds. Finally, in further support of the sensory hypothesis, DiPietro et al. (2008) found similar fetal responses to induced maternal relaxation as they did to induced maternal stress.

Specifically, when pregnant women in the 32nd gestational week underwent an 18-minute relaxation procedure, their heart rate and skin conductance levels decreased (opposite of their responses to the Stroop task) but, paradoxically, their fetuses showed some of the same responses as they did to the stressor task: a decrease in fetal movement and an increase in fetal heart rate variability. As before, there were only modest associations between changes in maternal physiology and fetal reactivity.

So far, with respect to the acute transmission of maternal psychological experience to the fetus, the data are affirmative: the fetus senses changes in the mother’s mood, but there is an unexpected twist. Mood-based changes in maternal physiology do not, for the most part, directly mediate fetal responses. Instead, the fetus senses and responds to changes in maternal experience via registering and reacting to changes in the mother’s autonomic nervous system activity.

**Contextual Influences on Fetal Development: Maternal Distress and Poverty**

As indicated, the question of the acute transmission of maternal experience to the fetus is inspired by and contributes to the meta question: do pregnant women’s psychological/psychosocial experiences shape the neurobiological development of the perinate, with implications for children’s long-term neurobehavioral trajectories? There is a burgeoning area of research suggesting that maternal anxiety, stress, and depression during pregnancy may have a negative impact on children’s psychosocial functioning (Charil, Laplante, Vaillancourt, & King, 2010; Talge, Neal, & Glover, 2007; but see DiPietro for contrasting findings: DiPietro, 2004; DiPietro, Novak, Costigan, Atella, & Reusing, 2006; DiPietro et al., 2010). With respect to psychosocial experiences such as poverty, there are extensive data documenting the negative impact of poverty on child development (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005), and some of it, beginning in utero, is focused largely on the quality of maternal nutrition (Georgieff, 2007; Georgiiff & Rao, 2001; Nelson, Wewerka, Borscheld, Deregnier, & Georgieff, 2003; Rao, Tkac, Townsend, Gruetter, & Georgieff, 2003). However, because the prenatal studies concerning maternal distress and poverty suggest a maternal influence during the in utero period, recent studies have aimed to identify a maternal effect at the time it is hypothesized to have occurred, that is, during pregnancy. More specifically, studies have aimed to find differences in fetal neurobehavior related to maternal distress and poverty.

In studies assessing fetal neurobehavior during a resting baseline period, several, largely consistent results have emerged. Greater life stress appraisal was associated with reduced fetal heart rate variability, indicating less optimal physiological regulation (DiPietro, Hodgson, Costigan, & Hilton, 1996c), as was increased movement, indicating heightened arousal (DiPietro, Hilton, Hawkins, Costigan, & Pressman, 2002). In another report, maternal depression, as assessed by a self-report index, also was associated with increased motor activity (Dieter et al.,...
2001; Emory & Dieter, 2006). Higher levels of maternal cortisol, a primary stress hormone sometimes found to be higher in the context of psychological distress, have been positively associated with greater amplitude in fetal movement, as well as with the amount of time fetuses spent moving (DiPietro, Kivlghan, Costigan, & Laudenslager, 2009). In contrast, relative to women who reported lower levels of trait anxiety, women who endorsed higher trait anxiety had fetuses who spent more time in quiet sleep and showed less gross body movement while in active sleep (Groome, Swiber, Bentz, Holland, & Atterbury, 1995). Although differences in fetal assessment may account for this seemingly conflicting finding (i.e., visualization of the fetus via ultrasound [Groome et al., 1995] versus computation of movement via digitized data from Doppler actigraphy [DiPietro et al., 2009]), it also likely reflects the relative newness of this scientific domain.

Other research has aimed to identify fetal differences related to maternal psychological functioning under conditions when the mother faces a challenge—the Stroop task, as described previously—as well as when the fetus is directly stimulated, that is, via a vibroacoustic probe. In the same studies discussed earlier, Monk and her colleagues found that whereas fetuses of nonanxious and psychically healthy women did not show a heart rate change during the Stroop task, fetuses of anxious and depressed women had a significant heart rate increase (Monk et al., 2000; 2004; 2010). In the most recent results, maternal cortisol levels were marginally positively associated with fetal heart rate activity (Monk et al., 2010). When stimulated directly with a vibroacoustic probe, fetuses of women with elevated levels of corticotropin-releasing hormone (CRH; which, during pregnancy, is predominantly of placental origin and can be increased by maternal cortisol) failed to respond to a novel stimulus in a habituation/dishabituation paradigm. In addition, higher CRH was positively related to higher overall fetal heart rate reactivity to the stimulus (Sandman et al., 1999). As is clear, embedded in this research is again the question of physiological mechanism: by what process is maternal psychological experience transmitted to the fetus in such a way as to alter fetal neurobehavior? More specifically, this is a search for a direct impact of women’s mood-based biology shaping fetal brain–behavior development. The hypothalamic-pituitary-adrenal (HPA) axis, particularly cortisol and its effects on brain development as well as placental protection against it; vasoconstriction in uterine blood flow; and, finally, cytokine activity are all being examined (Glover, Bergman, Sarkar, & O’Connor, 2008; Glover, O’Connor, & O’Donnell, 2009; Glover, Teixeira, Gitau, & Fisk, 1999; Wadhwa, 2005). Of note, in a few studies, DiPietro et al. have shown that greater fetal movement leads to higher levels of skin conductance (DiPietro, Caulfield, et al., 2006; DiPietro, Irizarry, Costigan, & Gurewitsch, 2004), which suggests that the direction of the maternal–fetal communication is far from fully understood, even as it relates to maternal states such as stress or depression. For example, could it be that more active fetuses contribute to greater sympathetic activation, which women interpret and “label” as a form of psychological distress (DiPietro, Costigan, Chen, & Voegtline, 2011)?

The effects of social class on fetal neurobehavioral also have been investigated, although the biology of these findings has been less considered. Poverty is associated with cumulative stressors (e.g., substandard housing, food insecurity, family turmoil, and community violence), and these sources of stress mitigate the association between low SES and poor developmental outcomes (Evans & English, 2002). The deleterious effects of lower SES extend into the prenatal period. Low SES has been associated with less fetal heart rate variability and less coupling of fetal movement and heart rate (DiPietro et al., 1999; Pressman et al., 1998). Intriguingly, in a study of pregnant women from Lima, Peru, and Baltimore, Maryland, DiPietro, Caulfield, et al. (2004) showed that fetuses in Lima had lower levels of resting heart rate variability and coupling, as well as a flatter slope representing the increase in coupling across gestation. Although both groups received prenatal care, the Lima group was less educated and, on average, 8 years older, and had less affluence and greater disadvantage. Nonetheless, this study failed to identify the maternal differences that could account for the fetal results, leaving the authors to underscore the role of the “broader context of the antenatal environment” in understanding maternal influences that may affect the fetus and, ultimately, the developing child.

**Conclusion: Fetal Neurobehavioral Development**

Although interest in fetal development, in the child before he or she can be seen, dates back to biblical times and beyond, recent advances in technology and, in particular, in knowledge of brain development before birth, have fostered a dramatic increase in studies aimed at characterizing what the fetus can do and by when. Overall, the findings on fetal state, as well as the maturational changes in heart rate and coupling, underscore the continuity between fetal and newborn behavior and the exaggerated divide that can be attributed to birth. Taken together,
these studies demonstrate that by the third trimester, it is possible to reliably “ask the fetus questions,” to use standardized assessment procedures and probes to evaluate neurobehavioral functioning that also can inform the identification of subtle individual differences in fetal development. However, a standardized battery for fetal neurobehavioral assessment does not yet exist, and most researchers follow DiPietro (2005) in cautioning that the production of such a standardized protocol is premature. Consistent with a renewed emphasis on the developmental origins of psychopathology (Bale et al., 2010; Insel & Wang, 2010), fetal neurobehavioral research also is focused on the factors contributing to individual differences, including variation in maternal mood-based biology. Similar to the research perspective on the postpartum baby, the fetus’ development is considered in the context of his or her antenatal environment.

The Infant

Here, we present research focusing on brain–behavior relationships that offers promise for examination of continuity from the fetal period and emphasizes basic processes in infant neurobehavioral growth. This focus on neurobehavioral development begins with an overview of neurological development and plasticity. We then review the literature on infant behavioral regulation, including state regulation and sleep. We review the work examining the dynamics of physical growth and motor development, including research on the mirror neuron system in infants. Our review of social-emotional development in infancy focuses on basic processes underlying key constructs such as attachment (Chapter 6, this volume) and includes a review of the infant’s emergent capacity to relate to others vis-à-vis attention regulation and sensitivity to social cues that extend from purely interpersonal exchanges to the eventual inclusion of objects. Although compartmentalized topically for the sake of organization, we have presented infant neurobehavioral growth as the product of dynamic and interrelated systems. This section on infancy culminates in a discussion of maternal depression and poverty, each of which illustrates the infant’s exquisite sensitivity to the broader ecology in which he is reared—from the “micro,” proximal level of the mother–infant dyad to the more “macro” or distal socioeconomic environment.

Infant Brain Development

Significant neurological development occurs in the first 2 years of life. At birth, the fully development neonatal brain weighs 400 g, approximately 25% of its eventual adult size. By age 2, the brain has advanced from 25% to 75% of its adult size, and this period of rapid growth is commonly referred to as the brain growth spurt (Schore, 1994). During this time, there is a maturational overproduction of axons, dendrites, and synapses across the cortex (Singer, 1995). Two primary mechanisms involved in functional neurological growth are synaptogenesis—the forging and strengthening of synapses that are in use; and synaptic pruning—the trimming of primitive synaptic connections that serve no functional purpose in the extraterine environment (Nelson & Bosquet, 2000). These two process are the central mechanisms of neural plasticity—the neural processes that underlies the degree to which experience shapes neural circuitry.

It has been suggested that there are two distinct mechanisms of plasticity (Greenough & Black, 1992). Experience-expectant development occurs only after the brain has received a certain critical threshold of input necessary for the formation of synapses that were not established prenatally. There are sensitive periods for which such input must be achieved in order for neurological change and its associated functionality to become instantiated. The most classic example involves the pioneering work of Wiesel and Hubel, who demonstrated that deprivation of light and form in kittens, via sutureing the eyes, led to blindness due to neurological changes to the visual system in the subcortical and cortical regions responsible for processing visual input. These effects showed little evidence of reversibility (Wiesel & Hubel, 1965) and were constrained to early life (Wiesel & Hubel, 1963), with no evidence of cortical blindness occurring in adult cats experiencing visual deprivation. Research examining the effects of cataracts on visual development in the human infant has showed that infantile cataracts are associated with long-term visual acuity (Birch & Stager, 1996) and ocular-motor (Abadi, Forster, & Lloyd, 2006) deficits. The deleterious impact of the cataract depends on the pathological characteristics of the cataracts (extent of visual deprivation) and the timing of surgical intervention—with significantly improved visual acuity (Birch & Stager, 1996) and ocular-motor functioning (Abadi et al., 2006) resulting when surgical intervention occurs within the first 6 weeks of life. More recent research has revealed that, following surgical intervention early in life, early visual deprivation associated with infantile cataracts leads to persisting deficits that are specific to human face processing (Robbins,
Nishimura, Mondloch, Lewis, & Maurer, 2010). Hence, early sensory and perceptual development is highly dependent on early experience and guided by experience-expectant processes, which are constrained by critical periods during which the brain must receive a requisite level of input.

The second form of plasticity is referred to as experience-dependent (Greenough & Black, 1992). This form of plasticity occurs when environmental input serves to create, strengthen, or modify synaptic connections. This process remains ongoing across the lifespan and is not as dramatically constrained by critical periods. Also in contrast to experience-expectant plasticity, which is generally specific to sensory and perceptual systems, experience-dependent processes are relevant for every domain of development—cognitive, behavioral, and social. Models of environmental deprivation have revealed the importance of the early rearing environment for cortical activity affecting multiple areas of functioning. In Romanian infants who experience extreme early deprivation (institutional care), brain activity is characterized by greater EEG power in low-frequency bandwidths of the EEG (θ) and reduced EEG power in high-frequency band widths (α and β), indicating slowed brain growth in children experiencing early deprivation (Marshall & Fox, 2004). Recent follow-up studies of institutionalized infants have revealed that environmental enrichment (foster care placement) is associated with increases in high-frequency EEG power (Marshall, Reeb, Fox, Nelson, & Zeana, 2008), and this improvement is influenced by duration of deprivation, with children experiencing enrichment prior to the age of 24 months showing significantly greater high-frequency EEG power than infants placed into foster care after age 24 months (Vanderwert, Marshall, Nelson, Zeana, & Fox, 2010). The neurological effects of early deprivation are further supported by behavioral and cognitive evidence. For instance, deficits in attention and visual processing in children who experienced prolonged institutionalization have been reported (Pollak et al., 2010). McLaughlin et al. (2010) reported significantly increased symptoms of attention deficit disorder in institutionally reared children and showed that this association was significantly mediated by reduced high-frequency EEG power and elevated low-frequency EEG power. The effects of environmental enrichment following deprivation are consistent with animal models. For instance, rodent models have shown that early exposure to enriched environments is associated with significant increase in the number of synapses per neuron in cortical regions involved in vision (Turner & Greenough, 1985).

Hence, neurological development in infancy is highly dependent on the quality of early experiences because such experiences facilitate the formation of synaptic connections that serve as the foundation for behavioral and cognitive growth.

The organization of the cortex continues across the first 3 years. Lateralization, or hemispheric specialization, is not consistently manifest as a stable, dominant hand preference until age 3 years (McManus, Sik, Cole, & Mellor, 1988). However, more recent studies have found evidence for stability in hand preference for reaching in infants between the ages of 7 and 13 months (Michel, Tyler, Ferre, & Sheu, 2006). This evidence is consistent with research using electrophysiological methodologies, which has provided evidence for emergent specialization of the left hemisphere for speech processing early in infancy (Dehaene-Lambertz & Balleit, 1998). Recent neuroimaging studies have revealed both structural and functional asymmetries in young infants. For instance, Glasel, Leroy, Dubois, Hertz-Pannier, Mangin, and Dehaene-Lambert (2011) showed a structural asymmetry in the right superior temporal sulcus of typically developing preverbal infants. Using functional neuroimaging, Dehaene-Lambert et al. (2010) found that maternal and novel speech samples activated the left temporal lobe in healthy 2-month-olds.

Research examining changes in hand preference has offered evidence that the development of lateralization is dynamic across the first 2 years and influenced by experience (Dehaene-Lambert et al., 2010). For instance, language milestones are associated with lateralization. Cochet, Jover, and Vauclair (2011) found that toddlers’ use of a consistent dominant hand for declarative pointing increased following the language growth spurt. Changes in handedness are also associated with motor milestones: prewalking infants showed decreased hand preference following the onset of hands-and-knees crawling (Corbetta & Thelen, 1999) and increased use of both hands, or coupling, in reaching following the onset of walking (Corbetta & Bojczyk, 2002). Taken together, this research indicates that there is evidence of lateralization present in infancy but that this process is dynamic across the first 3 years and influenced by linguistic and sensory-motor experiences.

There may be significant cognitive implications for the early development of lateralization across development. Consistency and type of hand preference in childhood has been of considerable interest in the etiology of dyslexia (Annett, 2011), and recent longitudinal evidence shows that, relative to children who did not show a consistent hand preference, girls, but not boys, showing a consistent hand preference from late infancy to early
childhood scored higher on tests of verbal intelligence and reading achievement in childhood and adolescence (Wilbourn, Gottfried, & Kee, 2011).

**Infant Behavioral States**

**Infant State Regulation**

The rapidly evolving organizational structure of early brain growth is evident in the behavioral organization of the neonate. At birth, the neonate’s sleep–wake cycle shifts more rapidly than that of a child or an adult. The neonate engages in patterns of motor activity that are similar to those observed during the late phase of the fetal period (Groome et al., 1999). The neonate’s sleep–wake cycles tend to show a modest degree of behavioral organization, and these cycles evolve rapidly across the first months. Newborns evidence 10 distinct behavioral states (Thoman & Whitney, 1990), as outlined in Table 1. The pace of cycling through these states slows as the infant’s behavioral organization becomes more predictable, with a gradual increase in the amount of time spent in waking states and sleeping schedules becoming more predictable by the age of 3 months (Henderson, France, Owens, & Blampied, 2010).

<table>
<thead>
<tr>
<th>State</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alert</td>
<td>Eyes open, attentive or scanning, gross-motor activity</td>
</tr>
<tr>
<td>Nonalert waking</td>
<td>Eyes open, attention unfocused, activity level variable</td>
</tr>
<tr>
<td>Fuss</td>
<td>Vocal distress sounds at low intensity, continuous or intermittent</td>
</tr>
<tr>
<td>Cry</td>
<td>Intense vocal distress, continuous or intermittent</td>
</tr>
<tr>
<td>Drowse</td>
<td>Eyes opening and closing or open narrowly, low activity level</td>
</tr>
<tr>
<td>Daze</td>
<td>Eyes open and glassy, no activity</td>
</tr>
<tr>
<td>Sleep–wake transition</td>
<td>Eyes may be open or closed, or vacillate between open and closed rapidly, generalized motor activity</td>
</tr>
<tr>
<td>Active sleep</td>
<td>Eyes closed, with REMs occurring intermittently, sporadic motor activity may occur; lowered muscle tone; emotion expression (smiling, crying) or other vocalizations may occur intermittently</td>
</tr>
<tr>
<td>Quiet sleep</td>
<td>Eyes closed, respiration slow and regular, motor activity limited and may include rhythmic mouthing</td>
</tr>
</tbody>
</table>

Adapted from Thoman & Whitney (1990).

State regulation serves as an important marker of self-regulation, as infants use shifts in state to adapt to the demands of their environment (Thoman, 1990). State regulation is an important marker of neurological and overall physiological functioning—shifts in state are associated with changes in cerebral metabolism and blood flow, endocrine function, and changes in cardiovascular and respiratory function (Orem & Barnes, 1980).

The emergence of prolonged periods of waking during the day allow for optimal levels of environmental stimulation, thus maximizing the opportunity for establishment of synaptic connections for emerging sensory-motor circuitry.
The increase in active alertness sets the stage for social experience. Cecchini, Baroni, Vito, and Lai (2011) showed that infant smiling varied during sleep and wake periods, with wakeful smiles showing clear links to social experience. The prolonged periods of active alert states provide opportunities for social communication, allowing caregivers to engage the infant in face-to-face play. Infant crying is also of social significance because vocalizing distress serves multiple functions, including communication and physiological regulation (Acebo & Thoman, 1995). Crying in response to hunger, fatigue, and discomfort offers an important mode of communication to caregivers who learn to differentiate these cries and respond contingently (Gustafson & Deconti, 1990). Style of maternal responding is related to infant crying—for instance, relative to lower frequencies of crying, higher frequencies of crying during maternal holding are associated with less interactional stability in the mother–infant dyad (Thoman, 1990).

Infant behavioral dysregulation is associated with early risk for mothers, infants, and dyadic functioning. Excessive crying in infancy is associated with maternal postpartum depression (Vik et al., 2009), lowered maternal perceptions of self-efficacy (Stifter & Bono, 1998), maternal report of physical and emotional symptoms and marital dissatisfaction (Levittzky & Cooper, 2000), and child abuse (Brewster et al., 1998). More generalized behavioral dysregulation is associated with other markers of early risk, including prenatal nutritional deprivation (Hernández-Martínez et al., 2011), maternal antenatal depression (Goodman, Rouse, Long, Ji, & Brand, 2011), prenatal drug and environmental toxin exposure (Myers et al., 2003; Stewart, Reihman, Lonky, Darvill, & Pagano, 2000), and premature birth (Stephens et al., 2010). In a large sample of 1,248 high-risk, prenatally drug-exposed infants, Liu et al. (2010) found that 5.8% of the sample showed extreme behavioral dysregulation in the neonatal period (i.e., highly aroused, hypertonic, low-quality movement, poor attention, and low self-regulation). These infants manifested persistent deficits on tests of mental development in infancy and cognitive and behavioral functioning in early childhood. These effects were independent of premature birth (gestational age) and low SES. Infants showing extreme behavioral dysregulation as neonates appeared to be the most sensitive to prenatal drug exposure and prematurity (Liu et al., 2010). The behavioral organization of the premature infant is also predictive of later health and behavioral difficulties (Ohgi et al., 2003; Stephens et al., 2010). In their sample of premature infants, Stephens et al. found that premature birth and a profile of underarousal on the Neonatal Intensive Care Unit Network Neurobehavioral Scales (NNNS; Lester & Tronick, 2004) predicted 26% of the variance in low motor performance on the Bayley Scales of Infant Development at 24 months. Ohgi et al. (2003) found that lower Neonatal Behavioral Assessment Scale (NBAS; Brazelton & Nugent, 1995) scores for motor and state control in very-low-birth weight (VLBW) neonates could be used to correctly classify 50–75% of children showing high levels of behavior problems in middle childhood (ages 7–8 years).

**Infant Sleep**

Sleep patterns in infancy change dramatically across the first year and are an important marker of behavioral regulation. Hormonal evidence of the emergence of circadian rhythms has been documented in 3-month-old infants (Rivkees, 2003). Behaviorally, consolidation of diurnal functioning occurs between months 4 and 6, when waking becomes consolidated to the day and sleep consolidated to night (Cornwell & Feigenbaum, 2006). These behavioral data are complemented by polygraphic data, which show high levels of variability in sleep states, including more frequent episodes of active sleep, prior to age 3 months (Hoppenbrouwers, Hodgman, Arakawa, & Geidel, 1988).

Like waking behavior, sleep patterns also become more organized across the early months of infancy. Using retrospective report methodology, Moore and Ucko (1957) reported that 70% of 3-month-old infants slept continuously for 5 consecutive hours (12 a.m. to 5 p.m.) by age 3 months. A more recent prospective longitudinal study examining parental sleep diaries of 75 typically developing infants between the ages 6 days and 12 months affirms the early consolidation of self-regulated sleep (Henderson et al., 2010). Henderson et al. noted that the most dramatic shift in increase in length of infants sleep occurred between the ages of 1 and 4 months, with the majority of infants sleeping for 8 consecutive hours by age 3 months and more than half the sample sleeping in accordance with family conventions (from 10 p.m. to 6 a.m.) by age 5 months.

Longer, more predictable windows of nocturnal sleep are also of considerable importance. Different sleep states appear to serve distinct neurobiological functions. For instance, in adults, non-rapid eye movement (NREM) sleep is associated with activation of preoptic and basal forebrain regions, and slowed respiration and activity supports...
energy conservation. In contrast, active (REM) sleep involves activation of the isolated brainstem and is characterized by a loss of muscle tone due to the simultaneous triggering of withdrawal and excitation motor neurons involved in memory consolidation and neuronal development. Neurological activity is high during REM sleep, and the proportion of REM sleep is highest in altricial species (where offspring are born immobile and heavily dependent on maternal care for survival), suggesting perhaps that activation of sensorimotor regions of the brain occurring during this time provides critical input necessary to support sensorimotor development (Siegel, 2005).

Additional evidence reveals that infants are capable of learning during sleep. Fifer et al. (2010) used an eye conditioning paradigm to examine infant contingency learning and associated neurological substrates in sleeping 1- and 2-day-old infants. Relative to infants who experienced random tone-air puff stimuli (control group), infants who experienced a consistent Pavlovian pairing of tone followed by air puff (experimental group) showed significantly more eye movement, specifically a fourfold increase in the likelihood of conditioned eye movements, in response to the presentation of a tone by the end of training. Event-related potential (ERP) data collected within the experimental group showed a more positive-going slow wave in the frontal regions, providing evidence of learning across trials in sleeping newborns. Another study applied similar eye-blink conditioning methodology in sleeping 1-month-olds assessed in the laboratory (Reeb-Sutherland et al., 2011). This study also examined differential learning during sleep based on type of unconditioned stimulus (social [female voice] or nonsocial [tone vs. backward voice]) and showed that learning across trials increased more rapidly for the social stimulus versus the two nonsocial conditions. Given the large proportion of time newborns spend in sleep states and the dramatic behavioral and cognitive gains of the first months of life, the need and capacity to learn likely extends to sleep (Fifer et al., 2010). Furthermore, social stimuli appear to hold a privileged position in early learning during sleep in the first month of infancy (Reeb-Sutherland, et al., 2011).

The evidence for infant learning during sleep states is complemented by research examining associations between sleep quality and developmental health. It is well-documented that persistent sleep disturbances in childhood are associated with poorer health, behavioral, language, and learning outcomes (Owens & Witmans, 2004; Quach, Hiscock, Canterford, & Wake, 2009). Evidence drawn from samples of children (Bourke et al., 2011) and preschoolers (Karpinski, Scullin, & Montgomery-Downs, 2008) has revealed that mild to severe levels of sleep-disturbed breathing are associated with poorer performance on tasks of executive functioning (Quach et al., 2009). Less is known about the long-term cognitive impact of disordered sleep in infancy. However, one study showed that a higher proportion of REM sleep (relative to total sleep time) is associated with lower developmental quotients in a sample of developmentally delayed infants (Shibagaki, Sawata, & Tachibana, 2004).

The quality of the early care environment affects infant sleep. Higher levels of maternal emotional availability (sensitive responsiveness tempered with appreciation for infants’ need for sleep [vs. stimulation] and the absence of hostility) during the bedtime routine was associated with less disrupted sleep in a sample of infants aged 1–24 months (Teti, Kim, Mayer, & Countermine, 2010). Morrell and Steele (2003) demonstrated continuity in sleep disturbances between the first and second year of life. Infants who showed a continuous profile of sleep problems were more likely than infants with no such stable profile of sleep difficulties to be rated as temperamentally difficult by their mothers and to have mothers who manifested problematic ideas about limit-setting and who relied on physical comforting at bedtime (cuddling or feeding to sleep; Morrell & Steele, 2003). The larger ecology of the family is an important contributor to quality of infant sleep. In a large sample of adopted children, Mannering et al. (2011) found that marital instability at infant age 9 months predicted infant sleep problems at 18 months. Another study of 35 infants found that typically developing infants with fathers who provided nighttime care woke fewer times during the night than did infants of less nocturnally involved fathers (Tikotzky, Sadeh, & Glickman-Gavrieli, 2011). Although these findings are correlational in nature, Tikotzky et al. suggested that more consolidated sleep for infants receiving high paternal care may be a function of more positive global co-parenting practices, which are associated with less parental stress and an enhanced familial atmosphere.

**Other Milestones in Infant Neurobehavioral Development**

**Motor Development**

The neonate is born with a set of motor reflexes that are markers of neurological health. Several of these reflexes have clear adaptive value and are known as survival reflexes. These include sucking, swallowing, and rooting, and...
collectively they reflect a set of hardwired motor milestones that serve to support survival (Wolff & Ferber, 1979). Of less obvious adaptive value are the primitive reflexes, which include the Babinski, Moro (startle), palmar grasp, stepping, and swimming reflexes. It has been suggested that these reflexes are lingering byproducts of our evolutionary history. Importantly, these reflexes are expected to be present at birth but disappear shortly thereafter (within 4–8 months postpartum). Indeed, the presence and expected departure of these reflexes are important markers of neurological health (Mac Neela, 1984).

Motor development is dramatic across the first 2 years as the sporadic undifferentiated movement of the newborn evolves into the impressive motor repertoire of the toddler. Like state regulation, there is evidence that generalized level of motor activity is stable from the fetal period to the first month of neonatal life (Groome et al., 1999). Action in infancy evolves rapidly and dynamically and is the product of the complex transactions between neurological growth, current level of motor functioning, physical and physiological functioning, and environment—the demands of the task at hand and the contextual forces (e.g., gravity) that impact performance (Thelen, 2005). A powerful example of this complex interplay is the demonstration that the “primitive” infant stepping reflex that disappears within 3 months following birth is readily apparent when the demands of the environment support infant stepping as the 3- to 8-month-old infant is held upright on a treadmill (Thelen, 1986). Such evidence was the first to demonstrate that motoric advances in infants are exquisitely catered to the demands of the environment but constrained by the concurrent developmental or physical state of the infant.

Support for the dynamic systems nature of motor development has been found in microgenetic studies examining change in behavior before and after the onset of a major motor milestone (see Table 2 for an overview of major motor milestones). For instance, infants who previously showed a one-handed preference in grasping will temporarily resort to bimodal grasping following the onset of sitting (Corbetta & Bojczyk, 2002), crawling (Corbetta & Thelen, 1999), and walking (Berger, Friedman, & Polis, 2011; Corbetta & Bojczyk, 2002). The onset of each new motor milestone requires the infant to learn new affordances for the new posture (Adolph, 2000). For instance, infants who have mastered sitting upright independently will not reach over a “risky” gap in surface to retrieve an object in a sitting position but will do so upon the onset of crawling when placed in the crawling position (Adolph, 2000). Although experienced crawlers will effectively judge and navigate a “risky” slope, this prudence is temporarily lost with the onset of walking, where the novice walker will proceed without caution (Adolph, 1997). The same may not be true for avoidance of drastic drop-offs. For example, new walkers showed more refusal when invited to cross to the deep end of a visual cliff than did experienced crawlers, even when placed in the more novel upright walking position (Witherington, Campos, Anderson, Lejeune, & Seah, 2005).
Table 2 Major Gross Motor Milestones from Birth Through Age 12 Months

<table>
<thead>
<tr>
<th>Motor Milestone</th>
<th>Mean Age (when 50% of infants have achieved milestone)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lifts head 90 degrees while on stomach</td>
<td>2.2 months</td>
</tr>
<tr>
<td>Rolls over</td>
<td>2.8 months</td>
</tr>
<tr>
<td>Sits with support</td>
<td>2.9 months</td>
</tr>
<tr>
<td>Sits without support</td>
<td>5.5 months</td>
</tr>
<tr>
<td>Stands with support</td>
<td>5.8 months</td>
</tr>
<tr>
<td>Crawling</td>
<td>7.0 months</td>
</tr>
<tr>
<td>Walks with support/cruising</td>
<td>9.2 months</td>
</tr>
<tr>
<td>Stands independently</td>
<td>11.5 months</td>
</tr>
<tr>
<td>Walks independently</td>
<td>12.1 months</td>
</tr>
<tr>
<td>Walks up steps</td>
<td>17.0 months</td>
</tr>
<tr>
<td>Kicks ball forward</td>
<td>20.0 months</td>
</tr>
</tbody>
</table>

Adapted from Bayley (1993).

Advancements in motor development influence other domains of development. Sitting independently allows for more effective object exploration (Soska, Adolph, & Johnson, 2010). The onset of walking is associated with increased use of distal objects and use of objects to initiate social interactions with caregivers (Karasik, Tamis-LeMonda, & Adolph, 2011). The onset of self-propelled movement is associated with the emergence of visual proprioception, or the visual awareness of one’s own movement, even when self-propelled movement is achieved artificially via training on a powered mobility device (Uchiyama et al., 2008). The onset of locomotion is associated with social-emotional changes in infants, including more positive affect and increased parental perceptions of both positive and negative affect (Whitney & Green, 2011).

The environment also influences infant motor development. Mothers use a rich communicative repertoire to assist infants in navigating their physical world (Karasik, Tamis-LeMonda, Adolph, & Dimitropoulou, 2008; Karasik, et al., 2011). Infants then use this social information to guide their locomotion (Adolph, Karasik, & Tamis-LeMonda, 2010), although this is tempered with their own perceptual judgments and increasing autonomy (Adolph et al., 2010). Use of infant walkers, which prevent the infant’s natural use of environmental input, is associated with slowed motor development (Garrett, McElroy, & Staines, 2002). Infants who experience less purposeful “tummy-time” or lower exposure to the prone (vs. supine) lying position (Majnemer & Barr, 2005) and overweight infants (Sloning, Adair, Goldman, Borja, & Bentley, 2010) may be at significant risk for delayed motor development, suggesting that nutrition and experience have important implications for motor development.

The development of complex action patterns in infancy is related to social experience. Observational learning is evident in the neonate’s ability to imitate simple facial gestures such as tongue protrusion and open-mouth facial gestures (Meltzoff & Moore, 1977; 1983). Neonatal imitation is also evident in nonhuman primates (Ferrari et al., 2009). Nine-month-old human infants have been shown to emulate novel, complex motor tasks via simple observation and can evidence deferred recall for complex novel actions, even generalizing those actions to manipulations with superficially different but functionally similar stimuli (Learmonth, Lambeth, & Rovee-Collier,
2004; Lukowski, Wiebe, & Bauer, 2009). Across infancy, observation of motor tasks is associated with mental representation of expectancies about the physical world. For instance, infants make predictions about the action intent of others, and these implicit representations grow increasingly more sophisticated across the first year, born from observation of others and their own accomplishment of motor milestones (Sommerville & Woodward, 2010). For example, infants look longer when the action of another ends in a manner that is incongruent with a previously demonstrated goal, such as when an adult experimenter who previously reached for one object switches her goal and grasps for a different object (Woodward, 1998). This association between action and representation is evident in actual imitation of action as well; 7-month-old infants selectively reproduce the goal-directed actions of others (Hamlin, Hallinan, & Woodward, 2008), and this appears to be specific to the actions of animate versus inanimate objects (Mahajan & Woodward, 2009).

An emergent body of evidence is revealing the neural substrates behind observational motor learning, elucidating the sophisticated implicit processes that underlie representations of complex motor tasks. Work with children (Lepage & Théret, 2007) has revealed that the frontal-parietal motor regions of the brain that activate while performing a goal-directed action also become activated when observing another perform the same action, a phenomenon referred to as motor resonance that has led to the suggestion of a mirror neuron system that underlies observational learning. Recent work with infants has revealed that such a mirror neuron system is in place early in life and likely plays a role in observational learning of complex goal-directed actions. Recent research using event-related EEG desynchronization has shown activation of the motor regions when 9-month-old infants executed or observed reaching and grasping (Southgate, Johnson, El Karoui, & Csibra, 2010). Marshall, Young, and Melzoff (2011) found that 14-month-olds showed activation of motor pathways while executing and observing a button-press task. This emerging body of research with infants provides new evidence to affirm the representational function of motor tasks, with the mirror neuron systems underlying the process by which social input is translated into action.

Social Development

Neonatal imitation and observational learning of motor tasks in infancy are clear demonstrations of the newborn’s exquisite sensitivity to the social world. Newborns show an immediate attunement to the social world. High-amplitude sucking methodology has revealed that infants prefer human speech versus nonspeech sounds, and this is evident in the first 1–4 days after birth (Vouloumanos & Werker, 2007). Additional research has shown that neonates look longer at face versus nonface stimuli in the first hour of life (Johnson, Dziurawiec, Ellis, & Morton, 1991). By 10–16 weeks, infants manifest a preference for viewing filmed social behavior that is synchronous (lip-voice synchrony) versus that which is temporally altered or asynchronous (Dodd, 1979).

Infants become increasingly more active participants in social exchanges with their caregivers across the first 4 months. Days after birth, infants manifest a visual preference for their mother’s face over that of a female stranger (Pascalis, de Schonen, Morton, & Deruelle, 1995). By age 6 weeks, infants coordinate their gaze behavior with maternal vocal behavior during face-to-face interactions (Crown, Feldstein, Jasnow, Beebe, & Jaffe, 2002). Examination of infant saccadic reaction times has revealed that, across the first 3 months, infant gaze behavior becomes increasingly more sophisticated, encompassing less fixated staring behavior (Hopkins & Van Wullfsten Pathe, 1985) and more deliberate scanning of targets (Hunnius & Geuze, 2004). More gaze control opens the door for more complex social coordination between infants and caregivers. Microanalytic coding of mother–infant interaction has revealed that 3-month-old infants coordinate their gaze (Harel, Gordon, Geva, & Feldman, 2011) and affect (Lester, Hoffman, & Brazelton, 1985; Tronick, 1989) to that of their mother. By 4 months, social coordination is evident in the vocal rhythms of the mother–infant dyad, and early patterns of infant vocal coordination with mother and stranger foretell social and cognitive outcomes at age 12 months (Jaffe, Beatrice, Stanley, Crown, & Jasnow, 2001). Asynchronous behavioral coordination with mother across multiple modalities, particularly facial-visual engagement and touch, at infant age 4 months is predictive of secure versus insecure attachment classification in toddlerhood (Beebe et al., 2010).

After the first quarter of the first year, the infant’s social exchanges with caregivers involve increased interest in their physical world. In the first 3–4 months, infants demonstrate the ability to follow the gaze of an adult toward a specific proximal target within their visual field (D’Entremont, Hains, & Muir, 1997) and disengage from the target to refocus their gaze on their caregiver (Perra & Gattis, 2010). By 6 months, infants follow more distal gazes of adults
but are likely to become fixated on distracters that enter their visual field before the distal target is identified visually (Butterworth & Jarrett, 1991). The visual precision of gaze following continues to evolve across the first year, such that between 12 and 18 months infants are able to follow an adult gaze to a target well outside of their direct visual field (Butterworth & Jarrett, 1991). Between 6 and 9 months, this attention following is evident by infant engagement in periods of joint attention, as the infant jointly shares attention to objects or other referents with others (Scaife & Bruner, 1975). Infants first manifest the ability to respond to bids for joint attention (Bakeman & Adamson, 1984) and eventually advance to initiating bouts of joint attention (e.g., via pointing at objects) by the age of 12-months (Tomasello, Carpenter, & Liszkowski, 2007). Joint attention continues to evolve across the second year as caregiver–infant interactions with objects gradually shift from maternal-driven to infant-led play with objects, in tandem with a shift from the infant’s reliance on gesture and physical play to the use of language (Aureli & Presaghi, 2010). This developmental shift from responding to bids for joint attention at 6 months to more initiation of joint attention by 12 months may be due to evolution in neurological advancement in neural circuitry involved in regulation of attention. Mundy, Card, and Fox (2000) acquired baseline EEG from infants at age 14 months and found a profile of parietal lobe activation (associated with posterior implicit attention regulation) was associated with the development of responding to joint attention bids between the ages of 14 and 18 months. Furthermore, development of initiation of joint attention between 14 and 18 months was associated with a profile of activation in the prefrontal and central pathways (involved in higher level, voluntary control of attention; Mundy et al., 2000).

The occurrence of joint attention between infants and their caregivers is associated with language learning because the shared attention to a common referent or object invites the opportunity to identify labels for referents (Bruner, 1975; Tomasello, 1992). Relative to mother–infant dyads who engage in lower frequencies of joint attention, dyads who engage in higher frequencies are characterized by more generalized levels of maternal verbal input during mother–infant interactions (Stevens, Blake, Vitale, & Macdonald, 1998). Infants who co-engaged in more frequent instances of joint attention with mothers at age 6 months had higher vocabulary scores in toddlerhood relative to infants who co-engaged in less frequent instances of joint attention (Saxon, 1997).

Like all dyadic processes, infants make their own important contributions to the social experience of shared attention. There are important individual differences in the degree to which infants can engage in joint attention or manifest joint attention skill (Mundy & Gomes, 1998). There is a positive association between infant skill in responding to bids for joint attention (gaze, pointing) and the frequency of joint attention episodes observed during mother–infant play (Markus, Mundy, Morales, Delgado, & Vale, 2000). Six-month-old infants (Morales, Mundy, & Rojas, 1998) and 12-month-olds (Markus et al., 2000) who more skillfully responded to joint attention bids than those who manifested less skill were reported to have higher vocabularies as toddlers. Individual differences in infant responding to joint attention at 12 months and initiating joint attention at 18 months were each positively associated with language at age 24 months (Mundy et al., 2007). Infants who respond to joint attention performed better on language at 18 months than infants who did not respond to joint attention (Brooks & Meltzoff, 2005).

It has been suggested that the experience of shared attention with another represents an important milestone in social cognition because engagement in joint attention represents the expression of the infant’s understanding of intentionality or the representation of other persons as intentional agents (Tomasello, 1995). Brooks and Meltzoff (2005) showed that 10- and 11-month-old infants were significantly more likely to follow the gaze of an adult when the adult’s eyes were open versus closed. The same was not true for 9-month-olds, who followed gaze regardless of whether the eyes were open or shut. Hence, although gaze-following is clearly evident in 6-month-old infants (Bakeman & Adamson, 1984; Butterworth & Jarrett, 1991; Scaife & Bruner, 1975), the representation of intention is only manifest months after the infant has engaged in attention regulation, suggesting that experience in attention regulation is a critical precursor to social cognition (Mundy & Newell, 2007). Clear links between joint attention in infancy and performance on subsequent measures of social cognition have been found. For example, joint attention skill in infancy is positively associated with toddler’s use of mental state language (e.g., expressions about their own cognitions, emotions, desires, perceptions, and physiological state; Kristen, Sodian, Thorner, & Perst, 2011), performance on theory of mind tasks (i.e., visual perspective-taking, “seeing-leads-to-knowing,” and a situation and desire-based emotion tasks; Charman et al., 2000), and parent report of global social competence in early childhood (Van Hecke et al., 2007).

**Emotional Development**
Emergent social information processing in infancy develops in tandem with the capacity to recognize emotions in others. Newborn infants look longer at a stimulus depicting a happy face versus a fearful one (Farroni, Menon, Rigato, & Johnson, 2007). Habituation studies have revealed that, by age 3 months, infants discriminate between faces depicting happiness versus sadness (Barrera & Maurer, 1981; Kuchuk, Vibbert, & Bornstein, 1986). In addition, a 3-month-old’s face recognition is better for affectively positive versus neutral faces (Turati, Montirosso, Brenna, Ferrara, & Borgatti, 2011). Discrimination of fearful faces comes online shortly thereafter. By age 5 months, infants discriminate between happy and fearful faces (Bornstein & Arterberry, 2003), and there is robust evidence that infants as young as 5 months respond with negative affect to the shift in maternal affect from normal face-to-face play to a “still-face” (Cohn & Tronick, 1983). By ages 4–7 months, infants show avoidance when presented with threatening versus nonthreatening faces (Hunnius, de Wit, Vrins, & von Hofsten, 2011). A corresponding neurological response indicating attentional arousal has been revealed in ERP studies of 7-month-olds viewing photographic images of fearful and angry faces (Hoehl & Striano, 2008). Infants are slower to habituate to fearful versus happy facial stimuli, suggesting that infants show an attentional bias to threat as young as 7 months (Nelson, Morse, & Leavitt, 1979). Additional eye tracking work with 7-month-olds has revealed that the preference to attend to fearful faces occurs in tandem with heart rate deceleration or a cardiac orienting response (Peltola, Leppänen, & Hietanen, 2011).

Toward the end of the first year, infants engage in social referencing, looking to the emotional expressions of others to determine how to approach novel people, objects, and situations (Campos, 1980). Research using social referencing paradigms has revealed that 10- to 12-month-old infants show wariness of a target that has been presented in the context of negative affective expressions in adults, even if those queues are depicted on a video monitor (Mumme & Fernald, 2003; Mumme, Fernald, & Herrera, 1996). This is complemented by emergent evidence using ERPs in younger infants; for instance, 6-month-olds show heightened attentional arousal when viewing a fearful (vs. neutral) face gazing directly at an object (Hoehl & Striano, 2010).

There is evidence that infant emotion processing is associated with social experience. Three-and-a-half-month-old infants discriminate between sad and happy facial expressions only when they are first habituated to these changes in affect (from sad to happy) as displayed by their parents (vs. a novel adult; Walker-Andrews, Krogh-Jespersen, Mayhew, & Coffield, 2011). Additional research has shown that social sensitivity to affectively positive faces at age 3 months is associated with their caregiving history; infants who were most sensitive (looked longer) to increases in the intensity of positive affect in still images had mothers who were observed during home-based interactions to focus the infant’s attention on them (mother) more frequently (Kuchuk et al., 1986).

**Contextual Influences on Infant Development: Maternal Distress and Poverty**

**Maternal Distress**

Healthy infant development depends on the quality of the postpartum environment. Research with institutionalized children has demonstrated that extreme neglect is associated with compromised physical, cognitive, and social-emotional development (Ghera et al., 2009; Marshall et al., 2008; Nelson et al., 2007). Less extreme perturbations in the quality of the early rearing environment are also of considerable importance to healthy development. For instance, low-quality, insensitive maternal behavior during routine care-focused tasks in infancy is associated with a neurobehavioral profile of stress reactivity and behavioral defensiveness (e.g., right frontal EEG asymmetry at baseline, inhibited behavior in the presence of an adult stranger, aggression during play with a novel peer, and maternal report of proneness to anger) that persists into early childhood (Hane & Fox, 2006; Hane, Henderson, Reeb-Sutherland, & Fox, 2010). Additional research shows that high-quality maternal behavior in infancy is associated with slower increases in fear reactivity from 4 to 16 months (Braungart-Rieker, Hill-Soderlund, & Karrass, 2010) and maternal report of fewer behavior problems and higher social competence in early childhood (Leerkes, Blankson, & O’Brien, 2009).

Sensitive maternal behavior also buffers against the deleterious effects of antenatal anxiety (Grant, McMahon, Reilly, & Austin, 2010; Kaplan, Evans, & Monk, 2008). For example, infants of mothers meeting criteria for diagnosis of anxiety or depression in the second trimester of pregnancy showed significantly higher basal cortisol (a neuroendocrine marker of heightened stress) than did infants of healthy women only when the quality of postnatal care at age 4 months was low (Kaplan et al., 2008). Women who suffer from antenatal depression and anxiety are
at significant risk for continued illness following the birth of their infant (Field, 2011; Joseffson, Berg, Nordin, & Sydsjo, 2001). Depressed mothers provide less stimulating home environments beginning early in the postnatal period (Conroy, Marks, Schacht, Davies, & Moran, 2010). A great deal of research has demonstrated that the quality of mother–infant interaction is significantly compromised in depressed mothers, which may take the form of withdrawn or intrusive interactive behavior (Tronick & Reck, 2009). For instance, mothers suffering from postpartum depression subsequently engaged their infants (at age 4 months) with less optimal levels of stimulation, including discordant attention co-regulation and attenuated gaze coordination, but heightened affect coordination (Beebe et al., 2008). This pattern of asynchronous social engagement between depressed mothers and their infants persists across the first year of infancy (Feldman et al., 2009). In toddlerhood, maternal depression is associated with less co-engagement in joint attention to objects (Goldsmith & Rogoff, 1997; Jameson, Gelfand, Kulcsar, & Teti, 1997).

The experience of being cared for by a depressed caregiver marks significant upheaval for the infant. Maternal depression distorts emotional communication in the mother–infant dyad and significantly alters the infant's social-emotional development (Tronick & Reck, 2009). The experience of maternal apathy and withdrawal is stressful for infants. Early in infancy, typically developing infants respond to a laboratory-induced experience of maternal depression (vis-a-vis the maternal “still-face” paradigm) with protest, gaze avoidance, and negative affect that carries over once a normative pattern of face-to-face play is resumed (Cohn & Tronick, 1983), suggesting that infants are sensitive to the expression of depressed behavior in their caregivers. The impact of depressed caregiving is evident in the behavior of infants of depressed mothers, who show a generalized profile of affective dysregulation, including more withdrawn, less active, and less affectively positive social behavior than do infants of nondepressed mothers; see Tronick & Reck (2009) and Field (2010) for reviews. This “depressed” pattern of interacting in infants of depressed mothers carries over to interactions with other adults in infants as young as 3 months (Field, Healy, Goldstein, & Perry, 1988). Relative to infants of nondepressed mothers, infants of depressed mothers evoke less positive behavior from unfamiliar adults (Tronick & Reck, 2009). When presented experimentally with the challenge of a maternal still-face in the laboratory, infants of depressed mothers (vs. infants of nondepressed mothers) show more reactive/active forms of touch behavior (Moszkowski et al., 2009). Relative to infants of healthy women, infants of depressed mothers show lower levels of social engagement, less self-regulation, and more negative affect (Feldman, et al., 2009). Infants of depressed mothers also show physiological differences consistent with stress reactivity and negative affect, including a profile of right frontal EEG asymmetry (for a review, see Field & Diego, 2008) and basal autonomic and neuroendocrine arousal (Diego et al., 2004).

Infants of depressed mothers continue to show a profile of risk across development. Postpartum depression is associated with insecure attachment (Campbell et al., 2004), attenuated social competence (Wu, Selig, Roberts, & Steele, 2011), compromised language development in early childhood (Brennan et al., 2000; Chapin & Altenhofen, 2010), and increased internalizing behavior problems in middle childhood (Anhalt, Telzrow, & Brown, 2007). Relative to children of nondepressed mothers, children who are reared by mothers who show a stable profile of depression across their childhood are significantly more likely to show behavior problems and engage in more risk-taking behavior in adolescence (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009).

Poverty

According to recent statistics, more than 1 in 5 children under the age of 5 live in poverty (Child Trends, 2012). Families with infants and young children are the most likely to dwell in poverty. For instance, in 2011, 25% of children 0–5 years lived in poverty, and 20% of children aged 5–17 lived in poor households (Child Trends, 2012). A large body of research has documented the multifaceted risk associated with poverty for infants and young children, including poorer cognitive, linguistic, social, and emotional functioning (see McLoyd, 1998, for a review). Less is known about the basic processes that underlie the association between poverty and poor developmental outcome—and this is in no small part due to the comorbidity of risk factors associated with poverty, including poor prenatal health care (Laditka, Laditka, & Probst, 2006), increased exposure to drugs in utero (Lester & Tronick, 1994; Liu et al., 2010), increased rates of maternal depression (Tandon, Perry, Mendelson, Kemp, & Leis, 2011; Rafferty, Griffin, & Robokos, 2010), low levels of paternal involvement (Fagan, Palkovitz, Roy, & Farrrie, 2009), and higher exposure to parental conflict (Forehand & Jones, 2003; Jones, Forehand, Dorsey, Foster, & Brody, 2005) and community violence (Forehand & Jones, 2003).
Poverty may directly influence early neurobehavioral development vis-à-vis deprivation of micronutrients. In families with infants and young children, limited consistent access to food, or food insecurity, is associated with lower levels of all varieties of foods, particularly fruits and vegetables (Kaiser et al., 2003). Infants and toddlers who experience food insecurity are at elevated risk for iron-deficiency anemia (Skalický et al., 2006). Iron-deficiency in infancy is associated with dysregulated sleep–wake cycles in infancy (Peirano et al., 2010) and slower activation of visual neural pathways in infants aged 6–24 months (Monga, Walia, Gandhi, Chandra, & Sharma, 2010).

Environmental lead exposure associated with unhealthy housing conditions may also place impoverished infants at a disproportionate risk for poorer neurobehavioral outcomes (Gump et al., 2007; 2009). In infants, exposure to lead is associated with increased cardiac and neuroendocrine reactivity to stress (Gump et al., 2007; 2009). The negative and cumulative impact of lead exposure is evident by early childhood. For instance, higher blood lead levels are associated with attention deficits in preschool children (Chiodo et al., 2007) and with disordered conduct (Markus et al., 2000) and lower intelligence (Chiodo et al., 2007) in childhood.

An emergent body of research is beginning to examine neurological differences in young children based on social class. In a sample of 5-year-olds, Farah and her colleagues examined neurocognitive profiles of low- versus middle-class children and found specific deficits for low-income children on tasks tapping the left/perisylvian/language systems and the prefrontal/executive system (Noble, Norman, & Farah, 2005). The evidence for language deficits in children of low SES is consistent with functional imaging research showing a positive association between SES and activation in the left inferior frontal gyrus in 5-year-old children during completion of a language task. Animal models are consistent with these data and highlight the mechanistic importance of the early rearing environment in terms of quality of postnatal maternal care (Caldji et al., 1998; Caldji, Diorio, & Meaney, 2000; Liu et al., 1997) and the level of enrichment of the home environment (van Praag, Kempermann, & Gage, 2000) on these effects (see Hackman, Farah, & Meaney, 2010, for a review). The mitigating role of early care environments is supported in the human literature. For instance, maternal engagement in infancy and not income-to-needs ratio predicted subsequent neuroendocrine dysregulation in a low-income sample of infants (Blair et al., 2008).

**Conclusion: Infant Neurobehavioral Development**

The impressive neurobehavioral growth realized across the first 2 years necessitates a prepared brain, an active infant, and a supportive environment—all of which act in concert to advance development. Early experience with self and others fuels neurobehavioral advancements in behavioral, motoric, and social and emotional development. Across infancy, there is complex interdependency across these systems. For instance, the capacity to imitate others is evident at birth (Meltzoff & Moore, 1977; 1983) and in mirror neuron activity (Marshall et al., 2011; Southgate et al., 2010). Moreover, the infant’s careful observation of the social world plays a central role in the development of action (Learmonth et al., 2004; Lukowski et al., 2009), representation (Hamlin et al., 2008; Mahajan & Woodward, 2009; Sommerville & Woodward, 2010; Woodward, 1998), and social information processing, including emotion processing (Walker-Andrews et al., 2011), social referencing (Campos, 1980; Mumme et al., 1996), and intentionality (Brooks & Meltzoff, 2005). Experience with movement is afforded by caregiver behavior (Garrett et al., 2002; Majnemer & Barr, 2005), and infant action itself informs perception (Adolph, 1997; 2000; Campos et al., 2000) and, in tandem with activation of higher level attentional mechanisms (Lepage & Théret, 2007), paves the way for the infant to begin initiating joint attention via use of gesture (Mundy et al., 2000). Given such interdependency, compromised environmental input that dampens any of these systems will likely result in costs to other systems. This is evident in the case of both maternal depression and poverty, each of which is associated with pervasive and persistent negative outcomes. Maternal depression affects infants’ basic capacity to read social cues (Tronick & Reck, 2009), and this is evident in the withdrawn behavior of the infant (Diego et al., 2004; Tronick & Reck, 2009) and subsequent low levels of social competence (Wu et al., 2011) and delayed language growth (Brennan et al., 2000; Chapin & Altenhofen, 2010) of infants of depressed mothers. Impoverished caregiving environments are associated with a host of poor outcomes across all domains of development (McLoyd, 1998) and neurological differences that are evident in childhood (Hackman et al., 2010) and that are likely mediated by the quality of early care environments (Blair et al., 2008; Hackman et al., 2010). In sum, healthy neurobehavioral development depends on a healthy, expectant postnatal brain; the continual behavioral input of the infant; and the environmental affordances necessary to accomplish each interdependent milestone.
From Fetus to Infant: Future Directions

In this chapter, we have reviewed the research on fetal and infant neurobehavioral development. Although to the extent possible we have reviewed research showing parallel domains of development, there is currently a dearth of research that demonstrates continuity from the pre- to postnatal periods. Expanding technology now allows researchers to “ask the fetus questions,” and this work clearly demonstrates fetal competencies and fetal sensitivity to context (i.e., the uterine environment). In the future, longitudinal research that makes use of parallel methods for assessment during pre- and postnatal periods will be critical. Longitudinal research that uses consistent methodologies moving from fetus to infant will elucidate trajectories to key outcomes that appropriately highlight ongoing person-by-environment transactions, which may or may not be interrupted by the event of birth itself.

For fetus and infant alike, development is shaped by the environment. Research on maternal depression and poverty cited herein converge to demonstrate this. Moving forward, research that extends the focus on mothers to the broader rearing ecology of the family system is warranted. Although historically understudied, recent research is amply highlighting the importance of fathers as “partners” in the prenatal period and as parents of infants. For example, paternal depression in the prenatal period is directly associated with infant irritability (van den Berg et al., 2009). Furthermore, lack of paternal involvement in the postpartum period is associated with increased severity in maternal postpartum depression (Séjourné, Vaslot, Beaume, Goutaudier, & Chabrol, 2012). Quality of father–infant interactions directly predicts subsequent behavioral development, with higher quality interactions foretelling fewer externalizing behavior problems (Ramchandani, Domoney, Sethna, Psychogiou, Vlachos, & Murray, 2013) and better social competence with strangers (Ferber, 2010) in toddlerhood. Despite this evidence, we know little about the mechanisms underlying paternal contributions to early development—for example, no study to date has examined the direct role of fathers in regulating neurological or physiological responding in infants. Fathers may also take a more “active” or “direct” role in shaping prenatal development because epigenetic evidence drawn from animals shows an association between paternal stress exposure before the time of conception and blunted HPA-axis responding to stress induction in offspring (Rodgers, Morgan, Bronson, Revello, & Bale, 2013). This effect may be mediated by epigenetic alterations to gametes, as male mice exposed to chronic stress prior to mating (vs. nonstressed experimental controls) showed differences in the microRNAs of their sperm (Rodgers et al., 2013). Hence, the role of fathers may not be limited to prenatal “support” systems, but instead include direct effects on antenatal development through epigenetic mechanisms. Future research must expand on the paternity literature, examining both the direct and indirect effects of paternal life history, mental health, and parenting practices on both fetal and infant neurobehavioral development.

In this chapter, we have provided an overview of the basic processes underlying early neurobehavioral development, while emphasizing the critical role of experience therein. Perhaps the most important extensions of this basic literature will be work that translates this information into applied programming. From conception on, early human development is marked by an emerging, expecting brain that depends on healthy environmental input. At no other point in development is there such opportunity to enrich—or restrict—neurobehavioral potential. There is clear value in maximizing environmental enrichment before birth. For example, in a small sample of subclinically stressed pregnant women, cognitive behavioral therapeutic intervention during pregnancy was associated with reduction in pregnant women’s waking cortisol levels following treatment (Richter et al., 2012). The stress-reducing effects of prenatal intervention on neuroendocrine functioning may persist after birth; Urizar and Muñoz (2011) found that a prenatal stress management intervention reduced cortisol levels in pregnant women at risk for depression (having a history of depression or actively depressed) at 6 and 18 months postpartum. Given associations between maternal antenatal stress and poorer birth and infant outcomes, such evidence points to the benefits of early intervention for both mother and fetus. Importantly, plasticity remains ongoing after birth, and additional research shows that prenatal programming can be modified by postnatal factors. For example, Kaplan’s work cited earlier showed that infants of depressed/anxious mothers only showed significantly higher basal cortisol (compared to infants of healthy women) when the quality of maternal postnatal care was low (Kaplan et al., 2008). Hence, pre- and postnatal factors operate in concert to shape development. Conceptualizing pre- and postnatal development with a sense of continuity, and with appreciation that both stages of development are characterized by complex and ongoing person-by-environment transactions, may allow the field to design prevention/intervention programming that is tailored for both content and timing in such a way that neurobehavioral potential is protected from compromise and/or enriched in a window of maximum opportunity.
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Notes:

(1) The term gestational age used herein includes the 2 weeks prior to conception, beginning with the first day of the menstrual cycle prior to conception.

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