Low Birthweight and Developmental Delays: Research Issues in Communication Sciences and Disorders

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Low birthweight (LBW), with or without prematurity, is a risk factor that is believed to contribute to developmental delays (DeVries & Dubowitz, 1985; Martikainen, 1992). As such, much research has been undertaken in an attempt to determine the etiology of LBW and assess its impact on a variety of infant-toddler behaviors. Definitions of LBW vary, but it is typically defined as a birthweight of less than 2500g (e.g., Virji, 1991). In some studies, however, different criteria for LBW are used (e.g., Drillien, Thomson, & Burgoyne, 1980). Because LBW systematically occurs in conjunction with prematurity, it is often difficult to investigate only full-term LBW babies when conducting research. The term “prematurity- (or premature-) LBW” will be operationalized here to refer to LBW babies who were born prematurely.

Causative factors of prematurity-LBW vary, ranging from prenatal environmental issues (e.g., maternal substance abuse) to genetic and chromosomal factors (e.g., Down Syndrome, sickle cell anemia) to maternal medical complications (e.g., toxemia). Indeed, Rosenblith and Sims-Knight (1985) explained that it is difficult to study the effects of prematurity-LBW because of the multitude of etiologies. Different causes may result in the same initial diagnoses of prematurity-LBW, but, as development progresses, they may manifest themselves in different ways. For the purpose of this article only, prenatal environmental factors will be addressed.

LOW BIRTHWEIGHT

Babies born with LBW, with or without prematurity, are considered “high risk” for developmental delays (Brooks-Gunn, Klebanov Kato, Liaw, & Spiker, 1993; Papile, Burstein, Burstein, & Koffler, 1978; Williams, Lewandowski, Coplan, & D'Eugenio, 1987). Baumeister (cited in Rojhaj et al., 1993) explained that LBW is considered a biological risk factor because it has been routinely associated with developmental delays. He cautioned that LBW may become a contributing factor for later developmental problems. Baumeister further reported that not only does LBW represent a risk factor for later neurodevelopmental problems (see also Rosenblith & Sims-Knight, 1985), but it also has been found to be related to “a multitude of precipitating conditions (e.g., low SES, low maternal education, teenage pregnancy)” (p. 703).

Prematurity may result in neurological impairments such as periventricular leukomalacia (Iida, Takashima, & Takeuchi, 1992) or intraventricular hemorrhaging (Levene, Fawer, & Lamont, 1982) and physical impairments such as hypotonia or cerebral palsy (e.g., DeVries, Eken, Pierrat,
Daniels, & Cesaer, 1992; Rosenblith & Sims-Knight, 1985). The lower the premature infant’s birthweight, the more significant the neurological impairment (Feldman, Evans, Brown, & Wareham, 1992; Williams et al., 1987).

Periventricular leukomalacia (PVL) is one of the most serious brain injuries that can be sustained, with the highest rate occurring in babies weighing less than 1000g at birth (Feldman et al., 1992). According to Feldman et al., PVL is “a sensitive prognostic indicator of adverse neurodevelopmental outcome” (p. 223). Children afflicted with PVL are at significant risk for visual problems, hearing impairment, and mental retardation (DeVries & Dubowitz, 1985).

In a study by Williams and colleagues (1987), between 30% and 65% of LBW infants (< 1500g) exhibited evidence of intraventricular hemorrhaging (IVH). Severe difficulties may result from grades III and IV hemorrhages, and mild mental or motor handicaps may result from grades I and II hemorrhages (Papile et al., 1978). Results of the study by Williams and colleagues revealed that the majority of children with IVH performed poorly on several developmental indices (e.g., verbal, perceptual, motor). Additionally, longitudinal data disclosed that many of the children with IVH experienced learning difficulties (e.g., short attention span), requiring placement in special education programs.

Concerning premature-LBW babies born with physical impairments such as hypotonia, delays in the areas of motor and/or speech-language development may occur. For example, a baby may not have the strength or control to sit, or an infant may be able to sit, but not be able to crawl or walk. The same baby may exhibit respiratory problems due to weak thoracic musculature and experience difficulty vocalizing because of an inability to coordinate respiration with vocalizations. Even a mild degree of hypotonia may result in some difficulties with oral-motor movement, thereby possibly resulting in delayed expressive language development (DeVries et al., 1992).

Shapiro, Palmer, and Capute (1987) presented a typical pattern for a child who is delayed in sitting or walking as a result of mild hypotonia. Once the early milestones of sitting and walking have been obtained, Shapiro and colleagues suggested that a delay in language development may be observed. They stated that such a child may demonstrate “non-handicapping cerebral palsy (CP),” but is not classified as CP because no physical therapy is required. They added, however, that this type of child is at an increased risk for other neurologic dysfunctions, resulting in further developmental delays. Premature-LBW infants, then, may not necessarily demonstrate concurrent delays; their delays may occur successively. Premature-LBW children have exhibited lower intelligence scores than normal birthweight children, with more pronounced differences during the early years of life (Breslau et al., 1994; Brooks-Gunn et al., 1993). Liaw and Brooks-Gunn (1993) examined scores on the Bayley Scales of Infant Development (Bayley, 1969) for premature-LBW children over a 3-year period and found five distinct cognitive developmental patterns.

Subjects were selected according to birthweight: higher birthweight (2001g–2500g) and lighter birthweight (< 2000g). Upon analysis, the cognitive patterns that emerged were as follows: (1) high scores, stable over time; (2) initially high scores that declined to average; (3) initially high scores that fell to below average; (4) average scores that fell to below average; and (5) low scores, stable over time. Scores for the middle three groups declined over the duration of the study, whether or not they received early intervention services. On further analysis, the group that obtained low scores and whose scores remained low consisted largely of children with very low birthweight (VLBW) (i.e., < 1500g).

Additionally, when the higher birthweight infants and toddlers were compared to the lighter birthweight children, scores from the former group were positively correlated with higher cognitive scores at each testing period. In other words, even though the scores of both groups declined over time, the scores of the subjects in the higher birthweight group were, on average, greater than the scores of the lighter birthweight subjects at each testing period. The lighter birthweight subjects were further subdivided into a VLBW group (< 1500g) and, as mentioned, this group consistently demonstrated the lowest cognitive scores. In response to the apparent decline of cognitive scores, Liaw and Brooks-Gunn (1993) postulated that this may be, in part, due to inflated scores at 12 months based on the outdated normative data on the Bayley Scales of Infant Development (Bayley, 1969).

Weisglas-Kuperus, Baerts, Smrkovsky, and Sauer (1993) cited several studies in which non-VLBW children demonstrated poor cognitive outcomes due to social factors. They explained that VLBW (< 1500g) is associated with “socio-demographic risk factors (e.g., single motherhood, poverty) which, in themselves, may be related to less favorable outcomes” (p. 658). Escalona (1982) suggested that because VLBW babies are already at biological risk, they may be even more vulnerable to social risk factors than normal babies. Bennet (cited in Weisglas-Kuperus et al., 1993) suggested that children at biological risk (such as the VLBW group) may have more difficulty coping in less advantageous social conditions.

Weisglas-Kuperus et al.’s (1993) study examining cognitive development of VLBW children revealed that at 1 year of age, biological factors (e.g., birthweight) were significant predictors of cognitive development. As the children became older, however, social factors appeared to assume a greater role in determining cognitive outcome and the role of biological factors decreased. Cognitive development in VLBW children from highly stimulating homes improved over the duration of the study, whereas the developmental scores of children from homes with little stimulation decreased. This indicated that even though the children were at high biological (and social) risk, they were not necessarily destined for poor cognitive outcomes (Schaeder, Rappaport, & Courtwright, 1987). Indeed, considerable research over the last 10 to 15 years has suggested that both biological and environmental (social) conditions play an important role in determining developmental outcomes (e.g., Liaw & Brooks-Gunn, 1993; Rojahn et al., 1993).

Achenbach, Howell, Aoki, and Rauh (1993) stated that as children become older, academic achievement relies more
on an individual’s cognitive abilities. In their study on the effects of early intervention on LBW children’s cognitive and achievement scores, they found that with increasing age, the discrepancy between the scores of LBW children who received intervention and those who did not increased considerably. At 4 years of age, socioeconomic status and intervention explained approximately the same amount of variance. At 9 years of age, however, they found little variance explained by socioeconomic status, and a higher percent of variance explained by the intervention procedures. They subsequently postulated that cognitive (and hence, academic) outcomes for older LBW children may be more affected by intervention than by social factors (e.g., SES).

Two problems related to determining the effects of prematurity-LBW were described by Rosenblith and Sims-Knight (1985).

- First, it is important to mention that the presence of short-term effects does not necessarily mean that there will always be lasting effects. Premature-LBW babies are definitely at risk for delays and need to be carefully monitored as they develop, but long-term developmental delays may not be inevitable.
- Second, it is difficult to determine developmental delays in very young children because few developmental milestones need to be reached in order for the child to be considered within normal limits (Kenny & Culbertson, 1993).

The majority of the literature concerning the assessment of premature-LBW children involves evaluations conducted between 1 and 3 years of age (e.g., Brooks-Gunn et al., 1993; Liaw & Brooks-Gunn, 1993). Rojahn et al. (1993) suggested that, except for severe handicaps, it is often difficult to predict developmental delays early in life. Others state that a delay in motor development may be identified, whereas a delay in language or cognitive development may not be evident until later (Brooks-Gunn et al., 1993; Kenny & Culbertson, 1993). Kenny and Culbertson (1993) supported this viewpoint by postulating that, whereas a variety of motor skills are operational during the first year, language does not truly develop until years 3 through 5. (By “language,” Kenny and Culbertson appear to mean syntactic development). However, others, such as Coplan, Gleason, Ryan, Burke, and Williams (1982) have shown that language deficits may be observed in children younger than 3 years of age (e.g., Ballot, Rothberg, & Katz, 1992; Janowsky & Nass, 1987).

Kenny and Culbertson (1993) provided another obstacle to detecting developmental delays during the preschool years—that of the wide range of acceptable variability in the acquisition of both motor skills and speech-language skills. For example, according to the Early Language Milestone Scale-2 (Coplan, 1993), emergence of the first word may occur anywhere from 9 months to 18 months without a delay being indicated.

Schrader et al. (1987) suggested that with increasing age, more accurate detection of such delays should be possible. They did not, however, suggest at what age better detection is possible. Veen, Ens-Dokkum, Schreader, Verloove-Vanhorick, Brand, and Ruys (1991) reasoned that “not all sequelae can be diagnosed at [two years]” (p. 33), to help substantiate why it was necessary to conduct a reassessment at 5 years of age. Some studies, as shown below, have indicated that premature-LBW children’s scores may improve with age (Hunt & Cooper, cited in Liaw & Brooks-Gunn, 1993). However, they, too, have only assessed through approximately age 5.

Veen et al. (1991) assessed nearly 1,000 premature and/or VLBW infants during a 5-year longitudinal study. Their findings indicated that the primary disabilities were due to neuromotor function, mental development, or speech-language development. When comparing assessment results obtained at 2 and 5 years of chronological age, Veen et al. found that there appeared to be a decrease in adverse outcome for these children. In contrast to several other studies, birthweight was not associated with outcome; however, gestational age was. Future research involves conducting a reassessment at 9 years of age to focus on cognitive, educational, and behavioral outcomes because, Veen et al. ascribe, many learning and behavioral problems may surface as the children become older.

A few investigations have examined school performance of 6- and 7-year-old LBW children (Drillien et al., 1980; Richman, cited in Rosenblith & Sims-Knight, 1985). Richman followed a group of premature-LBW infants who attained all developmental milestones within normal limits and revealed that 48% of these children displayed signs of speech problems at 6½ years of age as compared to 6% of the normal birthweight controls. Drillien et al. found that, at 6:8 (years:months), LBW children were different from non-LBW children in a variety of academic behaviors (e.g., reading and spelling).

In summary, research over the past 2 decades has demonstrated that developmental delays involving cognition, neuromotor function, and/or speech-language are likely to occur in LBW children. Delays may or may not become manifest at an early age and, if they are evident, it does not mean that the delays will be long-term. Similarly, if no delays are observed early on, it does not mean that the child will be free from later developing cognitive or neurological deficits. Two environmental factors that, over the years, have been proven to contribute significantly to low birthweight and, therefore, developmental delay, are smoking and alcohol consumption.

**Smoking and Alcohol as Contributing Factors**

Smoking and alcohol consumption are two primary environmental factors that occur prenatally and are highly related to low birthweight (e.g., Moore & Persaud, 1993). Studies investigating the effects of either one of these teratogens customarily have a considerable number in their subject pool who have been exposed to both smoke and alcohol. In order to best determine the effects of one teratogen, the second one is factored out (i.e., held constant) during the analysis. Numerous studies have actually revealed a synergistic effect of smoking and alcohol on low birthweight—“the combination of the two is worse than either alone” (Rosenblith & Sims-Knight, 1985, p. 129).
Smoking. The U.S. Department of Health, Education, and Welfare (cited in Rosenblith & Sims-Knight, 1985) reported that, in 1979, at least 45 studies had confirmed the relationship of smoking to low birthweight and prematurity for a variety of socioeconomic classes and races. Floyd, Zahniser, Gunter, and Kendrick (1991) ascertained that one in four babies is at risk for LBW because their mothers smoke, and, overall, 21% to 39% of LBW births may be attributed to smoking.

Intrauterine growth retardation has been established as the primary cause of LBW in babies of smokers (e.g., Moore & Persaud, 1993), and it has been found that LBW babies of smokers weigh approximately 200g less than babies of nonsmokers (Floyd et al., 1991; Rosenblith & Sims-Knight, 1985). Additionally, a “dose-response” relationship has been demonstrated—the more cigarettes smoked, the lower the birthweight (e.g., Floyd et al., 1991).

Rush and Kass (1972) suggested that low maternal weight gain associated with smoking impacts birthweight. They found that the more cigarettes a woman smoked, the less weight she gained, and lower maternal weight gain is associated with an LBW baby. Placental abnormalities associated with smoking may also contribute to low birthweight (Meyer, Jones, & Tonascia, 1976). It has been hypothesized that blood being supplied to a fetus from a smoking mother is poorly oxygenated, resulting in lower birthweight.

Although numerous studies have confirmed a relationship between smoking and low birthweight, they disagree on whether there are long-term adverse effects. Some investigations demonstrated that LBW children were below normal birthweight controls in reading level, school adjustment, and spatial orientation (Scott & Latchford, 1976), but these results failed to replicate in subsequent studies. One important confounding variable in studies that examined academic outcomes in relation to prenatal smoking is that the outcomes may have been influenced by postnatal experiences such as environmental or social factors (e.g., SES, maternal education) (Rosenblith & Sims-Knight, 1985).

Alcohol. Alcohol consumption, like smoking, leads to an increased risk of intrauterine growth retardation, which has been shown to be an etiologic factor in LBW (Moore & Persaud, 1993; Virji, 1991). As with smoking, a dose-related response has been noted (Rosenblith & Sims-Knight, 1985; Virji, 1991).

Mukherjee and Hodgen (cited in Rosenblith & Sims-Knight, 1985) conducted a study with monkeys who were in the third trimester of pregnancy. Via an incision through which the umbilical vessels could be viewed, they found that within 15 minutes of the mother receiving an injection of alcohol solutions, the fetus’ umbilical vein collapsed. Circulation gradually improved over the next hour, but until blood flow was normal, the fetus suffered from oxygen deprivation. Even though such an experiment is impossible to perform with humans, it is hypothesized that placental insufficiency does also affect human fetuses (Rosenblith & Sims-Knight, 1985), resulting in poorly oxygenated blood being delivered to the fetus. The lack of sufficient amounts of oxygen may lead to intrauterine growth retardation, among other medical complications (Ginsburg, Blacker, Abel, & Sokol, 1991).

Virji (1991) explored the relationship of alcohol intake and infant birthweight in a population of 5,400 mothers (i.e., drinkers and nondrinkers) who gave birth to LBW, singleton babies. Fourteen percent of nondrinkers’ babies were LBW and 33% of heavy drinkers’ babies were LBW, resulting in a significant difference between groups. The percent difference of LBW babies born to nondrinkers (14%) and moderate drinkers (17%) was also significant. Virji’s results confirm previous studies that demonstrate an adverse connection between moderate to heavy alcohol consumption and birthweight.

Conversely, a more recent investigation conducted by Verkerk, van Noord-Zaadstra, du Florey, de Jonge, and Verloove-Vanhorick (1993) found that alcohol intake was not related to birthweight. In this study, however, a subgroup of women who consumed a moderate amount of alcohol daily did have LBW babies if the women also smoked at least one pack of cigarettes a day. In their conclusions, Verkerk et al. stated that “the effect of moderate alcohol consumption on birthweight corrected for gestational age is limited” (p. 128).

When comparing the studies by Virji (1991) and Verkerk et al. (1993), it should be noted that “moderate drinkers” were classified differently in the two investigations. Verkerk et al. classified moderate drinkers as those who consumed between 51g and 120g of alcohol weekly (63.75 ml to 150 ml), whereas Virji’s moderate drinkers consumed between 7.5 ml and 231 ml weekly. Moderate drinkers in Virji’s study, therefore, may have consumed up to 80 ml more per week than moderate drinkers in the study by Verkerk et al., which is equivalent to approximately four to five additional drinks weekly.

A second difference in the investigations is that Virji’s was retrospective, whereas Verkerk et al.’s was prospective. All mothers in Verkerk et al.’s study were interviewed during their first and second trimester about their alcohol and smoking habits during pregnancy. Questions pertaining to alcohol intake and smoking during the last trimester were asked a few days after delivery. In Virji’s study, mothers were asked about their current drinking and smoking habits, and it apparently was inferred that these current habits were also in effect during their pregnancy. Importantly, however, the length of time between delivery and interview was not provided.

Contrary to the results of the study by Verkerk et al., though, it appears that the majority of investigations concerning alcohol consumption and birthweight do find a definite inverse relationship—the more alcohol consumed, the lower the birthweight. Verkerk et al.’s study does, however, reiterate what several researchers believe, that of the synergistic effects of combining alcohol with smoking.

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**RESEARCH ISSUES**

With continuous advances in medical technology, more premature-LBW babies are surviving (Touwen, 1986). A recent study, for example, revealed that 50% of infants weighing less than 750g at birth (i.e., extreme prematurity)
were surviving (Llaad-Bruno, Lilley, & Westby, 1993). Currently, neonates born as early as 21 weeks gestation have a chance of survival. However, these babies will not weigh much and may exhibit significant medical complications due to extremely prematurity and VLBW. With such premature neonates surviving because of new medical and technological advances (Schaeder et al., 1987), there is an ever-expanding pool to investigate in relation to LBW and developmental delay.

There are many research issues to be considered when conducting research on the LBW population. First and foremost, operational definitions for parameters such as (V)LBW must be determined a priori (i.e., Will LBW be considered < 2500g or < 2000g?). When comparisons are made between different studies on LBW children, often times, children of different birthweight are being compared (e.g., one study may use < 2500g as the criterion, whereas the comparative study uses < 1500g). According to Touwen (1986), there are internationally agreed on definitions of (V)LBW, although not all researchers adhere to the suggested guidelines. In order to be able to compare studies of (V)LBW populations, it would be best if investigators used common, accepted definitions for (V)LBW.

A second significant decision regarding the sample is whether VLBW children will be included in with LBW children, or will they comprise a separate group. Many studies on LBW children do not always differentiate between LBW and VLBW, and this makes it even more difficult to compare performance outcomes across studies. It has been shown that VLBW children typically exhibit poorer scores/outcomes than LBW children (e.g., Llaw & Brooks-Gunn, 1993). If an LBW group also contains VLBW children and no distinctions are made between the two, scores/outcomes may be lower for the LBW group than they would be if VLBW children were not included, thus introducing a confounding variable. Even when the range of acceptable birthweights has been operationalized, the sample may still be heterogeneous because of different gestational ages. Most studies, however, choose to define study populations primarily by birthweight, not gestational age.

To reduce the heterogeneity of an LBW sample resulting from a variety of gestational ages, one might choose to restrict the range of gestational ages included in the study. In other words, the final sample may consist of LBW children (2001g–2500g) with gestational ages between 30 and 35 weeks, or VLBW children (< 1500g) between 23 and 28 weeks gestation may be sampled. Although this would increase the homogeneity of the sample, it would also more than likely reduce the number of cases included in the study.

Generalizability of results is largely based on the sampling choices made. If the parameters chosen to define the sample population are too stringent (or if there are too many parameters), the study may be well-defined but lack generalizability. Not only will the data obtained only be generalizable to a very narrow population, but the sample also may not be realistically replicable. In order for the information gleaned from a study to be useful to researchers and practitioners, it is important that the design and methodology be well-defined. However, to have a study that is too strictly defined at the expense of generalizability and replicability does not appear to be practical, productive, or cost-effective.

An exception to this may occur if a new issue or concern regarding LBW infant-toddlers were to be addressed (e.g., the identification of a previously unassociated etiology for LBW or the examination of a heretofore unresearched behavioral outcome). In instances such as these, it may be necessary to have more stringent inclusion criteria, for example, so there will be as few confounding variables as possible, even at the possible expense of generalizability and replicability. If results appear favorable, the subject criteria could then be expanded to make conducting more widespread research plausible.

As evidenced by the many studies on short-term effects of LBW presented, more longitudinal studies need to be undertaken. Although it is not always feasible to conduct longitudinal studies, it is important that they be undertaken with the LBW population for several reasons:

- The presence of short-term effects does not necessarily mean that there will be long-term effects, and, conversely, just because no short-term effects are evident does not mean there will be no long-term effects.
- The majority of studies have examined short-term developmental outcomes through 3 or perhaps 5 years of age, before the child is taxed by academic demands.
- The few studies that have examined LBW children’s academic performance beyond the age of 5 have typically only assessed children up to age 7 years.
- With increasing age, as more cognitive and language demands are placed on the children, it is important for educators and interventionists to understand the abilities of children classified as (V)LBW.

**SUMMARY AND IMPLICATIONS FOR FUTURE RESEARCH**

In summary, research designs for examining the relationship between LBW infant-toddlers and developmental delay need to include explicit operational definitions for terms such as LBW and VLBW. In order to simplify comparative analyses of studies, the internationally agreed on birthweights should be implemented for these terms. Studies should state the range of birthweights being investigated, thereby allowing readers to determine if VLBW infants and toddlers are being included in the analyses. If VLBW children are included with the LBW population, and no distinction is being made between the two groups, readers need to exercise caution when interpreting results. For research results to be of practical importance to fellow researchers and practitioners, subject parameters must not be too narrow. Parameters should be chosen to limit the number of confounding variables present, yet need to be broad enough to enable replication studies and generalizability to the population at large.
LBW and VLBW children may differ in the language acquisition process, as well as in how they develop language. In order to best capture potential differences between the two groups of children, the groups need to be clearly defined and, at least initially, be as homogeneous as possible. Investigations that compare the acquisition and/or development of different aspects of language (e.g., semantics or pragmatics) in children of LBW and VLBW may indeed reveal differences in the way these communication components progress. Differences in language acquisition and language development between the two groups of children may suggest the need for different types of treatment plans.

Achenbach et al. (1993) reported that as children become older, cognitive abilities play a significant role in academic achievement. The ability of a child to understand and express him- or herself in school also is important for academic success. The need for studies that document (V)LBW children’s cognitive and linguistic abilities throughout the high school years is necessary because if it is demonstrated that these children exhibit difficulty in “catching up to” or “keeping up with” their age peers, monitoring of their academic progress is essential in order to help maximize their potential. These children may need ongoing support, perhaps in the form of in-class speech-language intervention that addresses the language needs of the curriculum. Studies of older (V)LBW children are also needed because delays are not always evident in the first 5 to 7 years of life. Examination of older children’s pragmatic abilities may reveal inappropriate pragmatic skills, and such deficits may hinder the development of social skills and self-esteem, both of which may impact school performance.

CONCLUSION

To date, literature on (V)LBW infants-toddlers has provided us with knowledge concerning possible adverse developmental outcomes that may be encountered. Although it is unknown whether short-term effects are temporary or permanent, we must be aware of their existence and provide services as needed. We must also be mindful that delays may appear as the children age, even if they did not appear to be present initially. Future research endeavors that focus on the performance outcomes of older school-age children will be beneficial by expanding our knowledge base concerning (V)LBW children.

REFERENCES


