



# Infant Body Composition

*The New Frontier in Pediatric Care*

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# Infant Body Composition

## *The New Frontier in Pediatric Care*

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### Effects of Body Composition on Infant Health

#### *Pediatric Disease States and Pre-maturity*

The prevalence of preterm births has steadily increased over the last two decades and has reached 12.5% of all births in the United States in 2004. Most of this increase has been in infants born between 32 to 36 weeks gestation. Compared with infants born at term, preterm infants are at greater risk of death and disability. Twenty percent of infants born before 32 weeks gestation and 1% of infants born 32 to 36 weeks gestation do not survive the first year of life<sup>1</sup>.

Although recent advances in medical technologies and Neonatal Intensive Care Unit (NICU) care have led to improved mortality rates, surviving infants are at significant risk of morbidity. The complications of preterm births arise from immature organ systems that are not yet prepared to support life in the extrauterine environment. These include pediatric diseases and physiological states at birth or shortly after birth, as well as diseases and conditions which develop at a later stage.

Short-term complications include Bronchopulmonary Dysplasia (BPD) that affect the respiratory system, Necrotizing Enterocolitis (NEC) and Short Bowel Syndrome (SBS) that affect the Gastro-Intestinal (GI) tract, and adverse neurodevelopmental outcomes. Preventing and managing these complications involves nutritional management of these infants. Nutritional practices centered on preventing and managing disease states are often not optimal and affect the body composition of the infants.

**NEC and SBS:** NEC is a gastrointestinal disease that predominantly affects premature infants. Premature infants have immature bowels that are unable to withstand the stress caused by the movement of food within the intestine and, therefore, allows bacteria to invade the intestinal walls and causes infection. To avoid the risk of NEC, at-risk infants are often fed exclusively through parenteral nutrition. This induces a significant reduction in GI mass which can lead to other complications<sup>2</sup>. Infants with Short Bowel Syndrome (Short Gut Syndrome) are unable to absorb nutrients due to anatomical and functional loss of parts of the intestine. Both conditions affect normal growth and body composition of the infant unless alternative feeding therapies are provided.

**BPD:** Studies have shown that preterm infants with BPD have impaired growth with a deficit in fat-free mass and total body fat at term when compared with healthy term infants<sup>3</sup>.

#### *Neurodevelopmental Outcomes*

Preterm infants who are Small for Gestational Age (SGA) are at risk for adverse neurodevelopmental outcomes when optimal nutrition is not provided. However, current practices are tailored to provide nutrition that has a high caloric value and is aimed at maintaining in-utero rates of growth, not quality of growth. While this practice has shown success in improving neurodevelopmental outcomes, these infants have higher adiposity levels at term (corrected age) when compared with full-term infants, and are at risk for metabolic syndrome later in life<sup>4</sup>. Current nutritional practices adopted to avoid motor impairment and adverse neurodevelopmental outcomes adversely affect the body composition of infants at risk.

#### *Expanding on the statement above: The neonatologist's dilemma*

Current feeding practices in preterm infants who are Adequate for Gestational Age (AGA) try to achieve in-utero rates of growth. In preterm infants who are SGA, in addition to achieving in-utero rates of growth, they try to provide for catch-up growth. While doing so, preterm infants have higher adiposity levels when compared with full-term infants at term-corrected age<sup>4, 6</sup>. Studies have also shown there are beneficial aspects to slower early growth<sup>5</sup>, which is also known as Beneficial Under-Nutrition (i.e., slower early growth is beneficial for long-term cardiovascular health).

In light of these findings, the neonatologist is faced with a dilemma: *Slower early growth and risk-adverse neurodevelopmental outcomes or allow for catch-up growth and risk long-term cardiovascular health?*

Identifying mechanisms that lead to increased adiposity in preterm infants will lead to a better understanding of the impact of the quality of nutrition provided and will ultimately help in developing a nutritional strategy that both improves neurodevelopmental outcome and minimizes long-term metabolic and cardiovascular adverse effects<sup>4</sup>

### **Maternal Obesity/Maternal Diabetes**

Recent animal studies have shown that in-utero exposure to maternal obesity increases the risk of obesity in offspring<sup>7</sup>. This confirms Barker's Hypothesis, otherwise known as the Fetal Origins Hypothesis. Results in animal studies have now been duplicated in human studies as well. Infants born to obese mothers were found to have a higher percent body fat when compared to infants born to mothers who were not obese<sup>8 (PP)</sup>. Another study showed that male infants born to obese mothers had a higher fat percentage when compared to male infants who were born to moderately lean mothers<sup>9(PP)</sup>.

Maternal obesity often leads to maternal diabetes. Increased exposure of the fetus to fuel sources leads to increases in fetal size and changes in body composition (fat stores increase)<sup>10</sup>. Studies have shown that infants born to mothers with gestational diabetes mellitus have increased fat percentages and decreased lean body mass percentages when compared with infants born to mothers with normal glucose tolerance levels<sup>11</sup>.

### **Breast Feeding Vs Formula Feeding**

Apart from the effects of human milk in decreasing infection, NEC rates, and decreasing childhood obesity, recent studies have shown there are significant differences in body composition between formula-fed and breast-fed infants. Formula-fed infants were found to have a higher fat percentage at one year of age when compared with breast-fed infants. Another recent study compared infants breast-fed exclusively from birth to six months and formula-fed infants. Significant body composition differences were found between breast-fed and formula-fed infants in the first six months of life<sup>12 (PP)</sup>.

It can be concluded that infant body composition is affected by the environment in-utero, as well as by extra-uterine factors.

In-utero factors that have been shown to affect body composition include genetic influences, maternal obesity, and maternal diabetes.

Extra-uterine (postnatal) factors that are known to affect body composition include gestational age at birth (preterm or full term), pediatric diseases, and alternative nutritional strategies adopted to prevent or as a result of pediatric diseases for at risk infants, and feeding practices adopted (formula or breastfeeding).

## **The Importance of Measuring Infant Body Composition**

It has now been established that there is an association between growth patterns in early life and the subsequent risk of the metabolic syndrome implicating early-life nutrition as the underlying mechanism<sup>13,14</sup>. The early life programming concept proposes that infant passes through critical windows of sensitivity or plasticity, during which environmental factors generate long-lasting variability in phenotype<sup>15</sup>. These critical windows include exposure of the fetus in-utero as well as in the early postnatal period.

### **Barker Hypothesis**

The Barker Hypothesis, otherwise known as the Fetal Origins Hypothesis, proposes that a small size at birth or in infancy is associated with an increased propensity to adverse health outcomes in adulthood. It is suggested that poor fetal nutrition induces fetal adaptations that program future propensity to adult disease (details on the exact mechanisms of programming does not fall into the scope of this effort, but can be found here<sup>16</sup>). However, small body size and weight at birth (or subsequently) has been seen as a marker of poor fetal nutrition. Studies have found a J-shaped or U-shaped relation between birth weight and the incidence of adult disease when categorized by BMI<sup>17</sup>. Similarly, in adults, the Body Mass Index (BMI) has a U-shaped association with mortality, whereas body fat has a linear association<sup>18</sup>. This relationship can be attributed to different relationships between mortality and fat mass, as opposed to lean mass. Both high levels of fat mass and low levels of lean mass are independently associated with poorer adult health outcomes<sup>19</sup>. Thus it is apparent that detailed information about body composition and not just birth weight would be a better indicator of later risk of adult disease.

### **Effect of Postnatal Growth**

There is some controversy over the strength of the effect of fetal programming in-utero versus postnatal periods. In some studies that found associations between low birth weight and later adult disease, the effects of low birth weight were not adjusted for later adult body size, suggesting that postnatal nutrition can play a part in the programming of adult disease as well<sup>20</sup>.

Postnatal weight gain has been found to have associations with programming of adult diseases in Western populations<sup>20</sup>. The risks for metabolic syndrome are greatest in those infants

born small, but subsequently gain the most weight<sup>13</sup>. Recent studies have also distinguished the contributions of different periods of postnatal weight gain to later phenotype and disease risk<sup>21</sup>. In another study<sup>22</sup>, faster infant growth was associated with greater central adiposity. In the same study, rapid infant growth was associated with indications of growth faltering in-utero, therefore confirming that associations between birth weight and later fat distribution are generated at least in part by the rate of postnatal growth, but are also associated with prenatal growth patterns.

In contrast to the effect of birth weight, the effect of infant weight gain on later body composition appears to differ systematically between industrialized and developing countries<sup>23</sup>. A study on UK children linked infant weight gain with adiposity, but not with lean mass<sup>23</sup>. In contrast, studies<sup>24,25</sup> from non-Western populations, indicate infant weight gain was linked with later lean mass, but not fat mass.

These effects of rapid infant growth are further amplified by fetal growth failure. Postnatal catch-up growth after fetal growth failure leads to increased adiposity.

Thus, it can be concluded that the programming effects of adult disease cannot be restricted to fetal programming or a certain period, but can be viewed as a series of critical windows where the environment and nutrition contribute to the risk of later adult disease. This series includes fetal programming, postnatal programming, and childhood growth.

## Current Measures of Infant Growth

### Reference Growth Charts

Current measures of growth referenced in the charts below include the following:

- *Weight*
- *Length (Height)*
- *OFC (Occipito-Frontal Head Circumference)*
- *Weight/Length or BMI*

#### 1. CDC Growth Charts, National Center for Health Statistics 2000

- *Weight, length, or height, OFC, weight/length, or BMI*
- *Male and female charts*
- *Birth-36 months and 2 years-18 months*
- *Needs correction for prematurity*

#### 2. Babson/Benda Intrauterine and Postnatal Growth Chart<sup>26</sup>

- *Combined sexes*
- *Built-in correction for prematurity*
- *Weight, length, OFC*
- *26 weeks – 12 months corrected age*

#### 3. Fenton Growth Chart<sup>27</sup>

- *Updated Babson/Benda*
- *Weight, length, OFC*
- *22 weeks GA to 10 weeks post-term*

#### 4. IHDP Growth Percentiles<sup>28,29,30</sup>

- *4 separate charts VLBW male/female and LBW male/female*
- *2 months to 38 months*
- *Weight, length, OFC*

Drawbacks and limitations of anthropometric indicators include:

Nutrition provided to preterm and term infants centers on achieving weight gain, length, weight/length, and head circumferences >10<sup>th</sup> percentile. Since there is currently no reference data on ideal body composition in preterm and term infants, the focus is on anthropometric indicators alone.

Anthropometric indices, while providing an important tool for assessing growth of infants, provide no information on the quality of growth achieved. The quality of growth achieved takes on a new importance with recent evidence pointing toward the effect of postnatal growth and nutrition to later risk to adult disease. Infants with the same weight/length measures can have differing adiposity levels. Qualifying nutrition to achieve optimum lean body mass and fat mass is important and required knowledge of infant body composition. Such qualification cannot be achieved with current growth measures.

## Clinical Uses of Infant Body Composition Information

### 1. Nutritional Intervention to ensure normal growth trajectory

Current feeding practices in the NICU target growth indices based on growth reference charts which do not include body composition information. It is also known that the quality of rapid growth is an important factor in the

early postnatal period. A body composition reference curve, used along with existing growth charts such as the CDC NCHS, Babson Benda, and Fenton charts, will help nutritionists adjust the micro and macro nutrient content of nutrition provided.

During the course of disease in infants, energy needs are affected by the underlying disease and current nutritional status. Some diseases have been shown to increase or decrease nutritional needs<sup>31</sup>. Body composition information can be used to determine the current nutritional status and will provide baseline information. Preterm infants recovering from pediatric disease states and complications like NEC, BPD, PDA, SBS, etc., have significantly altered body composition as a result of either malnutrition or alternate feeding therapies. During recovery it is important to calculate energy needs, as well as protein needs, to accrue new tissue to ensure growth<sup>31</sup>. Body composition data will be useful in calculating energy and protein requirements for these infants.

Nutrition to preterm infants who are SGA provides for catch-up growth and tries to maintain in-utero rates of growth. Early aggressive nutrition, while achieving the above, may cause higher adiposity levels in infants. Infant body composition can be monitored to see the effect of nutrition provided. Adjusting the protein and caloric content of nutrition provided to these infants based on body composition will ensure optimal growth without adding to the risk of metabolic syndrome in later adult life.

## 2. Use of body composition data to calculate dosage and treatment requirements

Current treatment requirements are tailored to body size and sometimes body surface area, as opposed to body composition. Examples include nutrition and fluid intake, drug dosages, radiation dosages, and dialysis. Pharmacological substances are ineffective at low concentrations and toxic at high concentrations<sup>19</sup>.

Most treatments and dosage requirements are currently based on body weight. The higher metabolic rate of infants is well recognized and, since over 99% of metabolic processes occur in lean mass, dosage calculations based on an infant's lean mass would be a more appropriate criterion in administering hydrophilic drugs<sup>32</sup>.

Fat mass may also be relevant in calculating anesthetic doses. Some anesthetic drugs are fat-soluble and absorbed into adipose tissue by diffusion. In obese individuals, the subsequent release of the drug from adipose tissue can delay recovery<sup>33</sup>. The effectiveness of anesthesia administration in the infant population using body composition data may improve clinical outcomes and needs more investigation<sup>19</sup>.

## 3. Optimizing NICU release criteria

Current NICU release criteria include, but are not limited to the following:

- *Consistent weight gain over a period of days*
- *Tolerating oral feedings*
- *Ability to maintain normal body temperature*

While consistent weight gain over a period of days is currently seen as a discharge criterion, qualifying the weight gain will help in identifying the infant's ability to maintain optimal body composition, and is even more important in preterm infants. Therefore, a better criterion would be to look at weight percentile together with body composition data.

In cases where the body composition of the infant deviates from the body composition reference curve, post-discharge nutritional recommendations can be made to achieve optimal body composition, while providing for adequate growth.

## 4. Develop normative growth data using body composition

Developing normative body composition data from subjects who are born full term and are breastfed will provide reference information as to how an infant is supposed to grow. This normative body composition data can be used to:

- *Refine nutrition guidelines*
- *Assess quality of growth in infants*
- *Assess nutritional status in regions and countries*
- *Add to current growth reference charts*
- *Develop better infant formulas*

## Infant Body Composition Measurement Technologies

### Densitometry (PEA POD)

This technique uses Air Displacement Plethysmography (ADP) via a device called the PEA POD<sup>®</sup>. An infant's body composition (fat and fat-free mass) are calculated from body density ( $\text{Density}_{\text{Body}} = \text{Mass}_{\text{Body}} / \text{Volume}_{\text{Body}}$ ). It is a complete turnkey system using the same patented air displacement technology as the BOD POD<sup>®</sup> Body Composition Tracking System, which has been used successfully for assessing the body composition of children and adults since 1994.

The PEA POD is extremely simple to operate, with software prompts guiding the operator through each step of the process. This includes inputting infant information into the software, weighing the infant, and measuring the infant's body volume inside the PEA POD chamber. Testing is completely safe and non-invasive, and there are no compliance issues. The temperature-controlled test chamber provides a comfortable test environment for the infant, and a complete analysis takes about 5 minutes.

Validation of the PEA POD has been performed against the deuterium dilution method and a reference 4-compartment model for the estimation of infant body composition<sup>34, 35</sup>. It was found to be accurate and precise, with excellent within-day and between-day reliability<sup>34</sup>. The ease of use, minimum safety concerns, and bedside accessibility, make the PEA POD highly suitable for monitoring changes in body composition during infant growth in research and clinical settings.

### Total Body Water (TBW)

This technique is based on the nutrition model of body composition. Since Total Body Water (TBW) is the main component of Fat Free Mass (FFM), if body water can be measured and the ratio of TBW to FFM is known, then Fat Mass (FM) can be calculated. To measure body water directly, an infant is administered a small amount of water labeled with deuterium, a non-radioactive tracer. Urine samples are collected before the oral dose and after several hours of the dose. Samples are analyzed using isotope-ratio mass spectrometry. Repeat measurements can be done only after the tracer from a previous measurement has cleared from the body, which is typically 10-14 days for infants<sup>34</sup>. The unavailability of the capability for mass spectrometry analysis in many labs, and the cumbersome nature of the procedure has not led to widespread use of this technology.

### Skinfold Calipers

This is a common method, typically performed using calipers that compress the skin at certain points on the body. While non-invasive, this method suffers from poor accuracy due to variations in fat patterning, misapplication of population-specific prediction equations, improper site identification for compressing the skin, poor fold grasping, and the necessity for significant technician training to administer the test properly. Also, no successful methodology for determining infant body composition using skinfold measurement has been devised.

### Bioelectrical Impedance (BIA)

BIA measurements rely on the fact that the body contains intracellular and extracellular fluids that conduct electricity by passing a high frequency electric current through the body. BIA determines body composition based on the body's measured impedance in passing the current and known impedance values for human muscle tissue. However, this method can be greatly affected by the hydration state of the subject and by variations in temperature of both the subject and the surrounding environment. BIA has not been successfully applied with infant subjects<sup>35</sup>. A recent study shows that BIA provides insignificant additional information when compared to anthropometry alone; i.e., infant weight was a better predictor of FFM when compared to the impedance index.

### Dual Energy X-Ray Absorptiometry (DXA)

DXA is a technique that was originally developed for determining bone mineral content in the detection and treatment of osteoporosis. More recently, application of the technique has been expanded to include the analysis of fat and lean mass of soft tissue, in addition to bone mass. In a study that compared DXA to a criterion 4-compartment model in adults, DXA body composition measurements were found to have a bias which varied according to the sex, size, fatness, and disease state of the subject<sup>38</sup>. Variations between software versions<sup>39</sup> (for subjects weighing < 40 kg) and manufacturers<sup>40</sup> have also been reported. Apart from studies questioning the accuracy and validity of DXA in the pediatric population, the testing procedure is affected by the following factors<sup>41</sup>:

#### 1) Operator-related issues

- Placement of external calibration standard
- Placement of blankets

#### 2) Subject-related issues

- Artifacts due to subject movement

## References:

1. Preterm Birth Book.
2. Special Circumstances: Trophic feeds, necrotizing enterocolitis and bronchopulmonary dysplasia. Reynolds RM, Thureen PJ. *Seminars in Fetal & Neonatal Medicine* (2007) 12, 64-70.
3. Growth and Body composition in preterm infants with bronchopulmonary dysplasia. Huysman WA, de Riddler M, de Bruin NC, van Helmond G, Terpstra N, Van Goudoever JB, Sauer PJJ. *Archives of Disease in Childhood Fetal and Neonatal Edition* 2003; 88:F46-F51.
4. Postnatal growth, neurodevelopment and altered adiposity after preterm birth – from a clinical nutrition perspective. Melinda Y. Yeung. *Acta Paediatrica* 2006; 95:909-917.
5. Is slower early growth beneficial for long-term cardiovascular health? Singhal A, Cole TJ, Fewtrell M, Deanfield J, Lucas A. *Circulation*. 2004;109:1108-1113.
6. Unpublished Data: Adiposity in Premature Infants at Term-Adjusted Age. Roggero P, Piemontese PM, Gianni ML, Orsi A, Amato O, Puricelli V, Mosca F. Presented at Pediatric Academic Societies Annual Meeting, San Francisco, May 2, 2006.
7. Maternal obesity at conception programs obesity in the offspring. Shanker A, Harrell A, Liu X, Gilchrist JM, Ronis MJ, Badger TM. *American Journal of Physiology. Regulatory, integrative and comparative physiology*. 2008 Feb; 294(2): R528-538.
8. Impact of maternal body mass index on neonate birthweight and body composition. Hull HR, Dinger MK, Knehans AW, Thompson DM, Fields DA. *American Journal of Obstetrics & Gynecology*. March 2008.
9. Maternal Body Composition is Related to Infant Body Composition, but only in males. Gilchrist JM, Andres A, Shankar K, Badger TM. Presented at International Symposium on In-Vivo Body Composition Studies. NY, USA.
10. Maternal Obesity, Metabolism, and Pregnancy Outcomes. Janet C King. *Annual Review of Nutrition*. August, 2006. Vol. 26:271-291.
11. Evaluation of body composition of large-for-gestational-age infants of women with gestational diabetes mellitus compared with women with normal glucose tolerance levels. Durnwald C, Huston-Presley L, Amini S, Catalano P. *American Journal of Obstetrics & Gynecology*, 2004 Vol. 191 No.3 804-808.
12. Unpublished Data: Body Composition Reference Data for Exclusively Breast-Fed Infants. Gilchrist JM. Presented at Pediatric Academic Societies Annual Meeting, May 5-7, 2007.
13. Fetal origins of adult disease: strength of effects and biological basis. Barker DJP, Eriksson JG, Forsen T, Osmond C. *Intl Journal of Epidemiology* 2002 31, 1235-1239.
14. The thrifty phenotype as an adaptive maternal effect. Wells JCK. *Biological Reviews* 2007 82, 143-172.
15. Programming by early nutrition in man. Lucas A. 1991. *The Childhood Environment and Adult Disease* pp. 38-55.
16. The developmental origins of adult disease (Barker) hypothesis. De Boo HA, Harding JE. *Australian and New Zealand Journal of Obstetrics and Gynaecology* 2006; 46: 4-14.
17. The influence of birth weight and intrauterine environment on adiposity and fat distribution in later life. *Intl Journal of Obesity* 2003; 27, 255-777.
18. Mortality associated with body fat, fat-free mass and body mass index among 60 year old Swedish men; a 22 year follow up study. *Intl Journal of Obesity*; 24:33-37.
19. Is body composition important for pediatricians? Wells JCK, Fewtrell MS. *Arch Dis Child*. 2008; 93: 168-172
20. Fetal origins of adult disease – the hypothesis revisited. Lucas A, Fewtrell MA, Cole TJ. *BMJ* 1999; 319:245-249.
21. Trajectories of growth among children who have coronary events as adults. Barker DJ, Osmond C, Forsen TJ, Kajantie E, Eriksson JG. *NEJM* 2005; 353, 1802-1809.
22. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. Ong KK, Ahmed ML, Emmett PM, Preece MA, Dunger DB. *British Medical Journal* 2000; 320, 967-971.
23. Programming of body composition by early growth and nutrition. Wells JCK, Chomtho S, Fewtrell MS. *Proceedings of the Nutrition Society* 2007; 66, 423-434.
24. Indices of whole-body and regional adiposity for evaluation the metabolic load of obesity. Wells JCK, Victora CG. *Intl Journal of Obesity* 2005; 29, 483-489.
25. Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurement of body mass index in childhood in the New Delhi birth cohort. Sachdev HS, Fall CH, Osmond C, Laskhmy R, Biswas SKD, Leary SD, Reddy KS, Barker DJ, Bhargava SK. *AJCN* 2005; 82, 456-466.
26. Growth graphs for the clinical assessment of infants of varying gestational age. Babson SG and Benda GI. *J Pediatr* 89: 815, 1976.
27. A new growth chart for preterm babies: Babson and Benda's chart updated with recent data and a new format. Fenton TR. *BMC Pediatrics* 2003; Vol 3:13.
28. Growth patterns of low birth weight preterm infants: longitudinal analysis of a large, varied sample. Casey PH,

- Kraemer HC, Bernbaum J, et al. *J Pediatr.* 1990;117:298–307.
29. Growth status and growth rates of a varied sample of low birth weight, preterm infants: longitudinal cohort from birth to three years of age. Casey PH, Kraemer HC, Bernbaum J, Yogman MW, Sells JC. *J Pediatr.* 1991;119:599–605.
  30. Weight-for-length reference data for preterm, low-birth-weight infants. Guo SS, Wholihan K, Roche AF, Chumlea WC, Casey PH. *Arch Pediatr Adolesc Med.* 1996;150:964–970.
  31. Energy. *Journal of Pediatric Gastroenterology and Nutrition* 2005; 41: S5-S11.
  32. The meaning and measurement of lean body mass. Roubenoff R, Kehayias JJ. *Nutr Rev* 1991; 49:163-175.
  33. Illustrations of inhaled anesthetic uptake, including intertissue diffusion to and from fat. Eger EI, Saidman LJ. *Anesth Analg* 2005; 100:1020-1033.
  34. Evaluation of body composition in neonates and infants. Ellis KJ. *Seminars in Fetal and Neonatal Medicine* 2007; 12, 87-91.
  35. Body composition of preterm infants measured during the first months of life: bioelectrical impedance provides insignificant additional information compared to anthropometry alone. Dung NQ, Fusch G, Armbrust S, Jochum F, Fusch C. *Eur Journl of Pediatrics.* 2007; Vol 166 No.3, 215-222.
  36. Review of DEXA in Pediatrics (LMI In-house review).
  37. DEXA measurements in small subjects: Conditions affecting clinical measurements. Koo WWK, Hockman EM, Hammami M. *Journal of American College of Nutrition,* 2004 Vol. 23, No. 3, 212-219.

## Company History

LMI was formed in 1989 after our founders recognized the importance of and need for a fast, accurate, safe, and easy-to-use system for assessing body composition. This led to the development of the company's first product, the BOD POD® Body Composition Tracking System, which was introduced in 1994.

Development of the BOD POD was supported by several Small Business Innovative Research (SBIR) grants awarded to LMI by the National Institutes of Health (NIH), the most prestigious health organization in the world. Since its inception, numerous research and trade publications have validated the accuracy, reliability, and ease-of-use of the BOD POD and its Air Displacement Plethysmography, now considered the "Practical Gold Standard" in body composition assessment.

Building upon the success of the BOD POD, LMI continues to introduce innovative and exciting new products with the same patented Air Displacement Plethysmography as the original BOD POD. Additional products include the BOD POD S/T (2002), PEA POD® Infant Body Composition System (2004), BOD POD *Gold Standard* (2007), and BOD POD *Express* (2007).

## Life Measurement Mission

The mission of Life Measurement, Inc. is to enhance human health and performance by providing the world's best products for measuring body composition.



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