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Energy expenditure, body composition, and disease risk in children and adolescents

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The purpose of the present paper is to review recent studies in children which have: (1) examined differences in energy expenditure, especially as they relate to differences in body composition and risk of developing obesity; (2) enabled more accurate measurements of body composition (especially fat and fat-free mass) and body fat distribution (especially intra-abdominal adipose tissue); (3) examined the relationship between obesity (especially body fat distribution) and increased disease risk.

ENERGY EXPENDITURE

Implementation of the doubly-labelled-water technique in human subjects in 1984 (Schoeller & Webb, 1984), and subsequent application in children has led to significant advances in our understanding of total energy expenditure in the paediatric population. Total energy expenditure has been measured in free-living children living in Phoenix, Arizona (Fontvieille *et al.* 1993), Burlington, Vermont (Goran *et al.* 1993*a*), Cambridge, UK (Prentice *et al.* 1988; Davies *et al.* 1994), and Belfast, Northern Ireland (Livingstone *et al.* 1992). The data are unanimous in showing that total energy expenditure in young children is approximately 25 % lower than current recommendations for energy intake (World Health Organization, 1985). This discrepancy may be explained by either: (1) inaccuracy of previous energy intake data used to derive the recommendations, or (2) a reduction in energy expenditure in children over the last few decades, presumably due to a decline in physical activity. Either way, these data imply that new nutritional guidelines need to be formulated for young children in order to ensure that recommended energy intake closely matches total energy expenditure. This may require reducing recommended levels of energy intake or recommending increased physical activity in order to reverse the recent cultural trends of reduced physical activity.

Studies of energy expenditure in children have also been useful for examining the role of energy expenditure in the development of obesity. Obesity arises from a failure in the regulation of energy balance leading to a mismatch between energy intake and energy expenditure, such that intake exceeds expenditure. The mechanism of this dysregulation is unknown, and it is not clear whether obesity develops because of an excess in energy intake relative to expenditure, a reduced energy expenditure relative to intake, or a combination of both. In adults, studies on the role of energy expenditure in the development of obesity have yielded inconsistent findings (Roberts *et al.* 1992; Seidell *et al.* 1992; Weinsier *et al.* 1993; Rising *et al.* 1994). Using the doubly-labelled-water technique, several cross-sectional studies in adults (Prentice *et al.* 1986; Lichtman *et al.* 1992; Welle *et al.* 1992), adolescents (Bandini *et al.* 1990), and children (Goran *et al.* 1995*b*; DeLany *et al.* 1996; M. S. Treuth, R. Figueroa-Colon, G. R. Hunter, R. L. Weinsier, N. F. Butte and M. I. Goran, unpublished results), as well as a meta-analysis (Carpenter *et al.* 1995), suggest that absolute total energy expenditure is higher in obese individuals, but is similar between lean and obese after normalizing for differences in body composition.

Several studies on the offspring of obese parents have been performed as these children may represent a model of the 'pre-obese' state. Using heart-rate monitoring, total energy expenditure was 22 % lower in eight children who had one or more parents with a history of obesity, compared with twelve children who had two parents with no history of obesity (4·9 (SD 1·2) MJ/d *v.* 6·3 (SD 1·5) MJ/d; $P < 0{\cdot}01$), even though the two groups of children were matched for weight and lean body mass (Griffiths & Payne, 1976). However, this study should be interpreted with caution because of small sample size and because the heart-rate method is unreliable in children (Livingstone *et al.* 1990). Also, the hypothesis that reduced energy expenditure is a risk factor for further weight gain was never demonstrated. In a later study reporting 12-year prospective data, energy expenditure failed to predict the development of obesity (Griffiths *et al.* 1987).

In infants born to either underweight (pre-pregnancy weight below the 10th percentile) or overweight (pre-pregnancy weight above the 90th percentile) mothers (Roberts *et al.* 1988), total energy expenditure at 3 months of age was 20% lower in six of the infants who became overweight after 1 year, compared with the remaining infants (255 (SD 27·1) kJ/kg per d *v.* 332 (SD 14·2) kJ/kg per d; $P < 0{\cdot}05$). Interestingly, this finding was refuted in a much larger study (*n* 124 infants) by other investigators from the same Institute (Davies *et al.* 1995) in which there were no significant correlations between total energy expenditure or sleeping metabolic rate in infants and maternal or paternal BMI. In the interpretation of the original study (Roberts *et al.* 1988), it is important to consider that the 'control' infants were born to severely undernourished mothers whose pre-pregnancy weights were less than the 10th percentile. Although this experimental design is useful for creating a paradigm that will maximize the chances of seeing an effect, an alternative explanation of the findings could be that energy expenditure is higher in babies born to undernourished mothers. Furthermore, there were only six infants with overweight mothers who gained excess weight in the first 3 months of life. This low sample size is a major concern given that two of the infants were outliers with an energy expenditure below the expected physiological range.

The effects of parental obesity on energy expenditure in offspring were examined in a cross-sectional study (Goran *et al.* 1995*b*) of seventy-three children (5 (SD 0·9) years: 20·4 (SD 3·8) kg). There were no significant correlations between any component of energy expenditure in children (total, activity, and postprandial resting) and body fat in children, nor body fat in mothers or fathers. In analysis of covariance (using fat-free mass as a covariate), there were no significant effects of gender in children, obesity in mothers, or obesity in fathers on total or activity-related energy expenditure in children (Goran *et al.* 1995*b*). However, there was a significant effect of gender and a significant interaction between obesity in mothers and obesity in fathers on resting energy expenditure in children (Goran *et al.* 1995*b*). Relative to children with two non-obese parents, resting energy expenditure was approximately 6 % lower in children when either mothers or fathers only were obese, but similar when both parents were obese (Goran *et al.* 1995*b*).

Several other cross-sectional studies suggest that energy expenditure is not abnormal in obese children. The influence of adiposity on energy expenditure components was examined by dividing forty-six children (boys and girls, African-American and Caucasian; aged approximately 10 years) into tertiles based on the sum of subscapular plus triceps skinfolds (DeLany *et al.* 1996). There were no significant differences across the three groups for any component of daily energy expenditure (total, resting, and activity). In Italian children (Maffeis *et al.* 1991) aged 8·8 (SD 0·3) years, those who were obese had similar values of resting energy expenditure to age-matched controls (4·9 (SD 0·39) *v.* 4·7 (SD 0·39) MJ/d), after correcting for differences in fat-free mass. Energy expenditure (24 h; in a metabolic chamber), total free-living energy expenditure (by doubly-labelled water), and body composition (by dual-energy X-ray absorptiometry (DXA)) were compared in twelve overweight (8·7 (SD 0·7) years; 46·5 (SD 9·0) kg) and twelve normal weight (8·2 (SD 1·0) years; 28·5 (SD 3·5) kg) children (Treuth *et al.* 1996*a*). BMR, sleeping metabolic rate, 24 h metabolic rate and total free-living energy expenditure were all significantly higher ($P < 0.01$) in the obese girls (by 0·93, 0·94, 2·17, and 1·82 MJ/d respectively). However, after adjusting for fat-free mass, all components of energy expenditure were similar in lean *v.* obese (e.g. adjusted means for lean *v.* obese were 6·2 (SE 0·29) *v.* 7·0 (SE 0·27) MJ/d for 24 h metabolic rate; 7·5 (SE 0·35) *v.* 7·5 (SE 0·35) MJ/d for total energy expenditure). These studies show that energy expenditure components by a variety of methods and under a variety of conditions are actually higher in overweight children, but this effect is explained by the greater fat-free mass. There is little evidence of reduced energy expenditure components in children with greater amounts of body fat.

Total energy expenditure and its components have been examined in other groups of children at high risk of developing obesity. In Mohawk Indian children, the prevalence of obesity is 44 % (Jackson, 1993). In a sub-group of these children who were matched for fat and fat-free mass to a group of Caucasian children living in Burlington, Vermont, total energy expenditure by doubly-labelled water, adjusted for fat-free mass, was significantly higher in the Mohawk children by 400–625 kJ/d (Goran *et al.* 1995*c*). In addition, resting energy expenditure has been shown to be normal in Pima children and in younger Mohawk children (4–7 years old) relative to Caucasian children (Fontvieille *et al.* 1992; Goran *et al.* 1995*c*). In Birmingham, Alabama, the prevalence of obesity (ideal body weight greater than 120 %) is higher in African-American (26 % in boys and 38 % in girls aged 10 years) compared with Caucasian (21 % in 10-year-old boys and girls) children (Figueroa-Colon *et al.* 1994). In thirty-five Caucasian children and sixty-four African-American children (Goran, unpublished results) there was no significant difference in total energy expenditure between genders or ethnic groups, after controlling for soft lean-tissue mass by DXA (adjusted mean values, 7·0 (SE 0·17) MJ/d in Caucasian *v.* 6·8 (SE 0·13) MJ/d in African-American children). Thus, reduced energy expenditure does not necessarily explain the greater prevalence of obesity in sub-groups of the paediatric population at greater risk of obesity.

Statistical techniques for data normalization are an important consideration in studies comparing groups of differing body size (Goran *et al.* 1995*a*). The first important issue is the selection of an appropriate covariate. As in adults (Ravussin *et al.* 1986; Weinsier *et al.* 1992), resting energy expenditure in children is most significantly correlated with fat-free mass (Maffeis *et al.* 1993; Goran *et al.* 1994). However, it is important to note that the regression slope between resting energy expenditure and fat-free mass decreases with increasing fat-free mass, probably due to an age-related increase in muscle mass:organ mass within fat-free mass (Weinsier *et al.* 1992). Thus, the relationship between energy expenditure and fat-free mass is not linear across all ages. The regression coefficient between resting energy expenditure and fat-free mass is 330 kJ/kg between age 0 and 2·5 years, 150 kJ/kg fat-free mass in 4–7-year-old children (Goran *et al.* 1994), 118 kJ/kg during adolescence, and 88 kJ/kg in adulthood (Weinsier *et al.* 1992). In addition, a gender difference in energy expenditure components should also be considered (Goran *et al.* 1994), as resting energy expenditure is significantly higher in boys, even after adjusting for fat-free mass (4·5 (SE 0·39) MJ/d), than in girls (4·3 (SE 0·32) MJ/d; $P = 0{\cdot}05$).

Another important data normalization issue is the use of ratios (i.e. dividing energy expenditure components by fat-free mass) *v.* regression (i.e. using fat-free mass as a covariate in an analysis of covariance). Ratios are frequently used even though historical (Tanner, 1949) and more recent (Ravussin & Bogardus, 1989; Toth *et al.* 1993) studies suggest they may lead to spurious data if the assumptions inherent in the ratio method are not met. The use of a ratio requires that the regression between the dependent variable and the covariate is linear and has a zero intercept, and these criteria are rarely satisfied (Tanner, 1949; Slaughter & Lohman, 1980; Ravussin & Bogardus, 1989; Goran *et al.* 1995*a*). Analysis of covariance, a regression-based approach, has been suggested as an alternative statistical technique to adjust data in this situation (Allison *et al.* 1995). We demonstrated the spurious nature of inappropriate data normalization in a previous study from our laboratory (Goran *et al.* 1995*b*). In this example we observed a significant inverse correlation between total energy expenditure (when adjusted for body weight) and fat mass in children (r −0·44; $P < 0{\cdot}001$). To demonstrate the spurious nature of this relationship we generated a random set of normally-distributed numbers with a mean and standard deviation equal to that of total energy expenditure in the data set (6·1 (SD 1·4) MJ/d). When the assigned random number was divided by actual body weight, a significant inverse relationship with fat mass was also observed (r −0.43; $P < 0{\cdot}001$). This phenomenon may explain why other studies which have normalized energy expenditure for body weight have observed an inverse relationship between energy expenditure and obesity (Roberts *et al.* 1992; Rising *et al.* 1994), including a recent review article (Schulz & Schoeller, 1994). Previously reported inverse relationships between energy expenditure and obesity may, therefore, be spurious and caution is warranted on the use of data normalization procedures in these types of studies.

Evidence, therefore, seems to be accumulating to refute the hypothesis that the development of obesity in children may be explained by a reduced level of energy expenditure and/or physical activity. Numerous studies, however, suggest that physical activity does play an important role in the regulation of body weight in children (Wilkinson *et al.* 1977; Dietz & Gortmaker, 1985; Bar-Or, 1993; Obarzanek *et al.* 1994). In attempting to reconcile this apparent paradox it is important to first recognize that there are many aspects of physical activity, and no real definition of the term exists. It is not known which of the various elements of physical activity are important in the regulation of body weight. Important factors to consider include intensity, activity time, metabolic efficiency, and the overall energy cost, as well as an appreciation of the type of physical activity (e.g. recreational, occupational, obligatory and spontaneous movement). Thus, several aspects of physical activity need to be considered, including quantitative (e.g. the energy cost) and more qualitative aspects (e.g. type and duration of activity), as well as the effects of exercise on intermediary metabolism (Poehlman, 1989; Forbes, 1991) and on appetite regulation (Kissileff *et al.* 1990).

The doubly-labelled-water technique provides an alternative non-invasive and unobtrusive measure of the energy cost of daily physical activity when combined with measures of resting energy expenditure. Physical activity energy expenditure derived in this manner, however, is not synonymous with physical activity *per se*. This is because activity-related energy expenditure derived by doubly-labelled water does not provide any information on physical activity pattern and does not discriminate the energy expenditure related to various types of physical activity (e.g. exercise, occupational-related activities, obligatory activities and spontaneous movements). Thus, physical activity energy expenditure may not always be representative of time spent exercising, because the daily energy expended in physical activity includes the combined energy cost of all physical activities including sedentary activities.

In previous studies which have demonstrated negative relationships between body fatness and physical activity (Wilkinson *et al.* 1977; Dietz & Gortmaker, 1985; Bar-Or, 1993; Obarzanek *et al.* 1994), it is not clear whether the negative influence of physical activity is mediated through the effects of activity energy expenditure or activity time. In a study of 101 pre-pubertal children, fat mass was inversely related to activity time (h/week) by questionnaire, but not activity energy expenditure in kJ/d by doubly-labelled water (Goran *et al.* 1997). These data suggest that obesity in children may be more related to time devoted to recreational activity rather than the combined daily energy expenditure related to physical activity. This finding implies that long bouts of physical activity (which can be sustained at low intensity) may be more protective than shorter bouts of high-intensity activity. Extended periods of low-intensity exercise may be more beneficial because of the promotion of an active lifestyle and, hence, the avoidance of a sedentary lifestyle implicated in promoting food intake, especially snacking (Taras *et al.* 1989). In addition, high-intensity activity does not ensure an increase in energy expenditure since it can cause a compensatory reduction in activity energy expenditure (increased sedentary activities) during the remainder of the day (Goran & Poehlman, 1992).

BODY COMPOSITION AND FAT DISTRIBUTION

Despite a recent surge of interest in body composition measurement techniques, relatively few studies have specifically addressed methodological aspects in younger children. Age-specific considerations are required since the usual assumptions in body composition models (e.g. hydration of fat-free mass, density of fat-free mass) are known to be influenced by age, and maturation state (Fomon *et al.* 1982; Slaughter *et al.* 1984; Lohman, 1986). Thus, age-specific equations for estimating body composition from skinfolds and hydrodensitometry have been developed (Lohman *et al.* 1984; Slaughter *et al.* 1984; Weststrate & Deurenberg, 1989). For skinfolds, prediction equations have been developed for children based on use of a multi-compartmental model combining measures of total body density (from underwater weight), total body water (from ^{2}H dilution), and bone mineral density (from photon absorptiometry) on the right and left radius and ulna as a criterion method (Slaughter *et al.* 1984).

Bioelectrical resistance is an alternative technique for assessing body composition in clinical and population-based studies. Age-specific equations have been recommended because age-related differences in electrolyte concentration in the extracellular space relative to the intracellular space may alter the relationship between bioelectrical resistance and total body water (Deurenberg *et al.* 1990). In a study that did not include adolescents, the relationship between height^{2}/resistance and total body water was robust across a wide age-range (Kushner *et al.* 1992). Moreover, the Kushner *et al.* (1992) equation has been cross-validated against total body water in 4–6-year-old children in two independent laboratories (Goran *et al.* 1993*b*) suggesting that bioelectrical resistance is not susceptible to inter-laboratory variation. Bioelectrical resistance has also been cross-validated in children against total body potassium (Schaefer *et al.* 1994). Bioelectrical resistance provides very reliable estimates of total body water; the intra-class reliability for estimates of fat and fat-free mass using bioelectrical resistance in twenty-six children (5·0 (SD 0·8) years; 20·2 (SD 3·0) kg) was > 0·99 for duplicate observations performed 2 weeks apart (Goran *et al.* 1993*b*). Collectively, these studies suggest that the Kushner *et al.* (1992) equation is valid across a wide age range, with the possible exception of adolescents.

Estimates of total body water by bioelectrical resistance can be transformed into equations for fat-free mass using published age- and gender-specific hydration constants (Fomon *et al.* 1982), although the original constants may have to be modified slightly (Goran *et al.* 1993*b*). Uncertainty exists over the exact hydration factor for fat-free mass and the factors contributing to its variability. One recent study used a combination of techniques to show that the hydration of fat-free mass was 72·7 % in pre-pubescent children (5–10 years), compared with 70·8 % in young adults (Hewitt *et al.* 1993).

More recently, DXA has been introduced as an alternative technique for assessment of total as well as regional body composition (Mazess *et al.* 1990). DXA is based on the exponential attenuation due to absorption by body tissues of photons emitted at two energy levels to resolve body weight into bone mineral, and lean and fat soft-tissue masses. DXA was originally introduced as dual-photon absorptiometry and used a ^{153}Gd radionuclide source to measure bone mineral density. Further developments led to the use of X-rays and reduced the radiation exposure to near background levels (approximately 0·04 mREM per scan; equivalent to several hours of background radiation at sea level), making the technique acceptable for research use in a paediatric population. Another advantage of DXA is the relatively quick scan time (20 min). Numerous studies have demonstrated good agreement between DXA and other laboratory-based techniques (Pritchard *et al.* 1992; Van Loan & Mayclin, 1992; Johansson *et al.* 1993), and four studies have examined accuracy by comparison against chemical analysis in animal models (Brunton *et al.* 1993; Svendsen *et al.* 1993*a*; Ellis *et al.* 1994; Pintauro *et al.* 1996).

In seven pigs (35–95 kg body weight; approximately 10–50 % body fat), DXA measures (using a Lunar DPX-L in the medium scan mode) were strongly related to carcass fat mass (*r* 0·99) and fat-free mass (*r* 0·98), and the regression lines relating DXA measures to chemical analysis were not significantly different from the line of identity (Svendsen *et al.* 1993*a*). The paediatric software for the Lunar DPX-L has also been validated against carcass analysis in pigs (Pintauro *et al.* 1996). In eighteen young pigs (16–36 kg body mass; 10–34 kg fat mass) the relationship between carcass lean content and DXA measures by the paediatric mode was highly significant (*r* 0·99), although the regression line was significantly different from the line of identity (slope 1·20, intercept −3·30 kg; SE of estimate (SEE) 3 % of lean mass). For fat mass, the relationship between carcass content and DXA measures was also highly significant (*r* 0·99) and significantly different from the line of identity (slope 0·87, intercept 0·19 kg; SEE 11 % of fat mass). In duplicate scans, the reliability of DXA measures of lean mass and fat mass was excellent (intra-class coefficients > 0·98; CV for fat mass was approximately 4 %, and the CV for lean mass was approximately 1 %). The deviations from the line of identity suggest that correction factors may need to be applied in order to improve the accuracy of fat and lean measures by DXA. Use of correction factors in this manner can effectively calibrate DXA to the laboratory standard of carcass analysis in a pig model (Pintauro *et al.* 1996).

The Hologic QDR-1000/W DXA instrument using paediatric whole-body software has also been cross-validated against chemical analysis (Brunton *et al.* 1993) in ten small (approximately 1·57 kg) and ten large (approximately 6 kg) piglets. In the small piglets there was modest agreement for lean tissue (*r* 0·92; significantly different from line of identity) and no agreement at all for fat tissue (*r* 0·06). In the larger piglets there was good agreement for lean tissue (r 0·96), and fat tissue (r 0·83), although in both cases there were departures from a 1:1 relationship. The Hologic QDR-2000 using adult scan analysis was examined in sixteen pigs weighing 5–35 kg (Ellis *et al.* 1994). There was a large discrepancy between DXA estimates of body fat and that measured by chemical analysis. The magnitude of the discrepancy was −1·16 kg fat mass (approximately 20% underestimate) for one software mode and ±0·75 kg fat mass (approximately 16% overestimate) for the second software mode. The regression line between chemically-derived fat mass and DXA-derived fat mass significantly deviated from the line of identity (slope 0·77, intercept 0·13 kg fat mass for one software mode; slope 1·1, intercept 0·31 kg fat mass for the other software mode).

Collectively, the validation studies of DXA suggest that the relationship between actual chemical content of the carcass and DXA estimates may be affected by factors such as the size of the animal, the equipment used, and the operation mode. These validation studies and the generation of new calibration equations are important steps in the development of standardized techniques to measure body composition in children (Pintauro *et al.* 1996).

DXA has subsequently been cross-validated (Goran *et al.* 1996) against skinfolds (using the Slaughter *et al.* (1984) equation based on triceps and calf skinfold measurements) and bioelectrical resistance (using the Kushner *et al.* (1992) equation and age-specific hydration constants). In forty-nine boys and forty-nine girls (6·6 (SD 1·4) years; 24·1 (SD 5·9 kg), fat mass by DXA (4·8 (SD 3·0) kg was significantly lower than fat mass by skinfolds (5·0 (SD 3·1) kg), although fat masses by these two techniques were strongly related (R^2 0·87; SEE 1·1 kg). Fat mass by DXA was also significantly lower than fat mass by bioelectrical resistance (5·7 (SD 3·4) kg), and the model R^2 (0·75) and the SEE (1·5 kg) were not as strong as for the skinfold technique. In forward regression analysis, subscapular skinfold, body weight, triceps skinfold, gender and height^2/resistance estimated fat mass by DXA with a model R^2 of 0·91 and an SEE of 0·94 kg fat mass, suggesting new anthropometric body composition prediction equations based on the use of DXA as a criterion method (Goran *et al.* 1996).

In adults, intra-abdominal adipose tissue (body fat around the visceral organs) is related to negative health outcome, independent of total body fat (Björntorp, 1992*a*,*b*). Thus, assessment of body fat distribution is equally as important as the measurement of total body fat. Traditionally, body fat distribution has been measured by anthropometry. Recently, *in vivo* imaging techniques (e.g. magnetic resonance imaging and computed tomography) have enabled more accurate measures of body fat distribution in children and adolescents. In sixteen lean and obese children (4–7 years), abdominal subcutaneous adipose tissue area was 6530 (SD 4480) mm^2, and intra-abdominal adipose tissue area was 830 (SD 580) mm^2 (Goran *et al.* 1995*d*). In a larger group of seventy-four African American and Caucasian lean and obese children (7·5 (SD 1·7) years; 33·0 (SD 12·2) kg body weight; 30 (SD 11)% body fat; Goran, unpublished results), intra-abdominal adipose tissue averaged 3000 (SD 2300) mm^2 but varied greatly (600–10200 mm^2); subcutaneous adipose tissue averaged 10100 (SD 9500) mm^2 (range 800–37200 mm^2). Intra-abdominal adipose tissue area was significantly correlated with subcutaneous adipose tissue area (r 0·83), total fat mass by DXA (r 0·79), soft lean-tissue mass by DXA (r 0·62), and body weight (r 0·77), but not age (r 0·33). Using magnetic-resonance imaging, in 11- and 13-year-old girls, intra-abdominal adipose tissue at the level of the umbilicus was 2410 (SD 410) and 2570 (SD 410) mm^2 respectively (de Ridder *et al.* 1992*a*). In a study in 11 year olds, intra-abdominal adipose tissue was 1780 (SD 1000) and 2480 (SD 880) mm^2 in boys and girls respectively (Fox *et al.* 1993). These values compare with typical values of

10000–12000 mm^2 in healthy adults (Lemieux *et al.* 1993), although it is difficult to compare absolute levels of intra-abdominal adipose tissue in adults with those of children because of differences in body size.

Waist:hip and waist circumference are often used in adults as markers of intra-abdominal adipose tissue. However, in children (Goran *et al.* 1995*d*) and adolescents (de Ridder *et al.* 1992*a*; Fox *et al.* 1993) there is no significant correlation between these markers and intra-abdominal adipose tissue as measured by imaging techniques. In children and adolescents, central skinfold thicknesses only explain 25–60 % of the variation in intra-abdominal adipose tissue (de Ridder *et al.* 1992*a*; Fox *et al.* 1993; Goran, *et al.* 1995*d*). Collectively, these studies confirm that circumferences may not be good indices of body fat distribution, whereas individual measurements of skinfold in the trunk region may be more useful. The use of DXA to measure total abdominal fat may provide a stronger index, but this technique cannot resolve subcutaneous from intra-abdominal adipose tissue. The combination of total abdominal fat by DXA and skinfold and/or anthropometry data (as an index of subcutaneous fat) has been used in adults to estimate intra-abdominal adipose tissue with reasonable accuracy (Svendsen *et al.* 1993*b*; Treuth *et al.* 1995*a*).

There is relatively little information on the factors accounting for differences in fat distribution in children and adolescents. Total fat mass is an important determinant of intra-abdominal adipose tissue, and in comparative studies data should be adjusted for total fat mass (Lemieux *et al.* 1993). However, only a portion of the variance in intra-abdominal adipose tissue is explained by total fat mass; in 206 women, the correlation was 0·75 (Treuth *et al.* 1995*a*), similar to that seen in pre-pubertal children (Goran, unpublished results). In obese (12·8 (SD 1·9) years; 70·6 (SD 15·4) kg) *v.* control (12·3 (SD 1·9) years; 38·4 (SD 7·2) kg) children, the increase in adipose tissue is predominately found subcutaneously (35300 (SD 9400) mm^2 *v.* 7900 (SD 6100) mm^2), although there is still an increase in intra-abdominal adipose tissue in the obese (4900 (SD 2100) mm^2 *v.* 2200 (SD 1100) mm^2).

With regards to sex differences, men have greater amounts of intra-abdominal adipose tissue than women, even after taking differences in total body fat into account (Lemieux *et al.* 1993). The gender difference in intra-abdominal fat is apparent, at least on an absolute basis, during adolescence (Fox *et al.* 1993), but not during childhood (Goran *et al.* 1995*d*). Since sex hormones are known to affect regional fat deposition (Björntorp *et al.* 1990), hormonal environment may contribute to sex differences in fat distribution that emerge after adolescence (de Ridder *et al.* 1992*b*). The hormonal environment plays a key role in determining body fat distribution, and this has been reviewed recently (Björntorp, 1996).

Ethnic background is one other factor that is known to affect fat distribution in children (Greaves *et al.* 1989; Goran *et al.* 1995*c*). Greaves *et al.* (1989) showed that black Americans and Mexican-Americans had greater fat in the central region by skinfolds, and that this held true for both parents and children. When Mohawk Indian children were matched in total body fat content to a group of Caucasian children (using bioelectrical impedance to measure body composition), subcutaneous fat (by skinfold) was more centrally distributed in the Mohawk children (Goran *et al.* 1995*c*). Since previous studies of ethnic differences in fat distribution have been limited to skinfold data, it is not known whether the findings represent differences in intra-abdominal or subcutaneous adipose tissue. In eighteen obese, adult females (Conway *et al.* 1995) African-Americans had lower intra-abdominal adipose tissue than Caucasians (10500 (SD 2500) mm^2 *v.* 1600 (SD 7000) mm^2), even though the two groups were matched for age, weight and total body fat. In children there are no published studies that we are aware of that have compared intra-abdominal adipose tissue among different ethnic groups using imaging techniques. Preliminary findings of Yanovski (unpublished results) suggest lower absolute intra-abdominal adipose tissue in African-American (*n* 21) compared with Caucasian (*n* 23) girls (7–10 years), although this difference was not significant when expressed relative to total body fat. In girls, preliminary data from our laboratory show that intra-abdominal adipose tissue was significantly lower in African-American compared with Caucasian subjects by 40 % (4500 *v.* 3200 mm^2), even after adjusting for subcutaneous abdominal adipose tissue, total fat mass; this finding held true in a sub-set of obese girls with >10 kg fat mass (5700 *v.* 4400 mm^2 for intra-abdominal adipose tissue adjusted for subcutaneous adipose tissue). Thus, evidence does suggest that intra-abdominal adipose tissue may be lower in African-Americans across the life-span. However, the important issue (in terms of health risk) is whether ethnicity influences the strength and/or magnitude of the relationships between intra-abdominal adipose tissue and the subsequent development of disease risk factors.

Finally, physical activity is an important determinant of intra-abdominal adipose tissue. In adults, physical inactivity is associated with elevated intra-abdominal adipose tissue (Seidell, 1991; Troisi *et al.* 1991; Björntorp, 1992*a*), and intra-abdominal adipose tissue can be selectively reduced after aerobic- (Schwartz *et al.* 1991) and strength-training exercise (Treuth *et al.* 1995*b*). In adults, the effect of physical inactivity on cardiovascular disease risk factors is mediated in part through the effects of activity on intra-abdominal adipose tissue (Hunter *et al.* 1996). To our knowledge, only one study in children or adolescents has examined the inter-relationships among physical activity, intra-abdominal adipose tissue and increased risk of disease using sophisticated measurement techniques. In an intervention study in obese girls, strength training slowed the increase in intra-abdominal adipose tissue, but not other compartments of body fat (M. S. Treuth, G. R. Hunter, R. Figueroa-Colon and M. I. Goran, unpublished results).

OBESITY, FAT DISTRIBUTION AND DISEASE RISK IN CHILDREN

The association between obesity and disease risk is well established. In adults, intra-abdominal adipose tissue has emerged as the specific fat depot associated with independent disease risk, including non-insulin-dependent diabetes mellitus and cardiovascular disease (Björntorp, 1992*a*). The associations among obesity, impaired insulin sensitivity and dyslipidaemia are reviewed elsewhere (Reaven, 1988; Frayn, 1995). However, the mechanism(s) and pathway(s) relating obesity, insulin sensitivity and dyslipidaemia are unknown. For example, it is unknown whether insulin resistance precedes and causes dyslipidaemia, or vice versa. It has been postulated that increased triacylglycerol turnover in intra-abdominal adipose tissue causes excess hepatic exposure to free fatty acids, which may increase hepatic gluconeogenesis and secretion of LDL, inhibit hepatic clearance of insulin, leading to hyperinsulinaemia and peripheral insulin resistance (Björntorp, 1992*b*). In prospective studies, fasting hyperinsulinaemia is a risk factor for future metabolic abnormalities (Haffner *et al.* 1992), and for ischaemic heart disease in men, independent of altered lipid levels (Després *et al.* 1996). In addition high fasting insulin and altered lipids may act in synergy with regard to risk of heart disease (Després *et al.* 1996). Most notably elevated fasting insulin (> 15 μU/ml) and apolipoprotein B concentration (>1190 mg/l) increased the odds ratio for the development of heart disease over time to 11·0 (Després *et al.* 1996).

On the other hand, some work suggests that inherent abnormalities in fat oxidation in the obese state cause changes in insulin sensitivity (Felber *et al.* 1993). According to the Randle *et al.* (1963) hypothesis, an increase in fat oxidation reduces the need for glucose

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oxidation leading to reduced glucose uptake and insulin resistance. However, there is no good evidence suggesting an inherent abnormality in fat oxidation in the obese state. Some studies support an alteration in fat oxidation due to obesity (Schutz *et al.* 1992; Astrup *et al.* 1994),while others do not (Lillioja *et al.* 1986; Calles-Escandon *et al.* 1995). Nagy *et al.* (1996) examined data from 720 healthy adult men and women. Fat oxidation was positively correlated with fat-free mass (*r* 0·32 in men; *r* 0·27 in women) and negatively correlated with fat mass in men (*r* – 0·19), but not women. After adjusting fat oxidation for fat-free mass, there was no significant relationship with fat mass. In healthy girls aged 7–10 years (M. S. Treuth, R. Figueroa-Colon, G. R. Hunter, R. L. Weinsier, N. F. Butte and M. I. Goran, unpublished results), fat oxidation over 24 h in a respiration chamber was higher in an obese sub-group, but this effect disappeared after adjusting for body composition. The lack of influence of fat mass on fat oxidation questions the hypothesis that altered fat oxidation plays a central role in modifying insulin sensitivity.

Relatively few studies have examined the relationship between early development of obesity and intra-abdominal adipose tissue and long-term disease risk. Data from the Bogalusa Heart study have shown a weak correlation between central body fat by skinfolds and insulin level in children (Freedman *et al.* 1987). Using DXA to measure percentage body fat (Gutin *et al.* 1994), the correlation between percentage body fat and fasting insulin in 10 year olds is 0·78 (Spearman rank correlation). In 8–11-year-old boys, insulin and the insulin:glucose were significantly higher in obese *v.* control subjects during a 3 h oral glucose tolerance test (Legido *et al.* 1989). Studies from France suggest that an abnormal pattern of insulin response to a meal is one of the earliest metabolic alterations characterizing the obese state, and that impaired insulin sensitivity is more apparent as the duration of obesity increases (Le Stunff & Bougnères, 1994). In one other study, obese children (59·4 (SD 2·3)% body fat; 11·6 (SD 0·6) years) had normal fasting insulin and 2 h oral glucose tolerance, but impaired peripheral insulin sensitivity (Monti *et al.* 1995). Collectively, most studies, but not all, suggest that one of the early responses to obesity is an increase in circulating insulin levels, and there is some evidence to suggest that this in turn may lead to the development of insulin resistance as the duration of obesity increases. Further longitudinal studies are needed to examine how early changes in insulin resistance track with obesity and whether these changes are the cause of the development of other metabolic abnormalities.

There are many cross-sectional and some longitudinal studies that have examined the relationship between obesity, body fat distribution and cardiovascular disease risk factors in the paediatric population (Siervogel *et al.* 1982; Shear *et al.* 1987; Freedman *et al.* 1989; Weststrate *et al.* 1989; Rolland-Cachera *et al.* 1990; Sangi *et al.* 1992; Durant *et al.* 1993). However, most of the previous studies are limited to cross-sectional analysis, and in studies that have demonstrated a link between body fat and cardiovascular risk, the correlations are weak (*r* values 0·1–0·3). The weak correlations may be due to the fact that body fat and/or fat distribution have usually been estimated from crude anthropometric indices. Using DXA to measure total body fat (Gutin *et al.* 1994) correlations of approximately 0·4–0·5 have been reported between percentage body fat and blood lipid levels in 10 year olds. In addition, using imaging techniques to measure fat distribution, intra-abdominal adipose tissue is significantly related to negative risk factors in obese children (e.g. total cholesterol *r* 0·54, LDL-cholesterol *r* 0·60, triacylglycerol *r* 0·46; insulin areas after an oral glucose test *r* 0·44), whereas no such relationships were seen for subcutaneous adipose tissue (Brambilla *et al.* 1994). Thus, the accumulation of fat in the intra-abdominal region is significantly related to adverse health effects, including dyslipidaemia and glucose intolerance.

SUMMARY

Recent methodological advances have led to a tremendous improvement in our ability to measure energy expenditure, body composition and fat distribution in children. The availability of new and improved measurement techniques has greatly enhanced the scope of research studies in children. The key findings from the present review are as follows: total energy expenditure in young children is approximately 25 % lower than current recommendations for energy intake and revised recommendations are necessary; reduced energy expenditure, however, does not necessarily explain the greater prevalence of obesity in the population as a whole or in sub-groups at greater risk of obesity; qualitative aspects of physical activity (e.g. time, intensity) may be more important than the energy expenditure of physical activity in the regulation of body composition; for body composition assessment, DXA is emerging as a technique which can substantially improve the accuracy and standardization in children; body fat begins to accumulate in the intra-abdominal region in young children and this accumulation is more pronounced in the obese; waist:hip ratio or waist circumference are inadequate markers of intra-abdominal adipose tissue in children and adolescents; finally, the early accumulation of fat in the intra-abdominal region is significantly related to the development of adverse health effects, including dyslipidaemia and glucose intolerance.

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