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ESSENTIALS Nutritional requirements for healthy pregnancy vary according to a woman’s pre-pregnancy nutritional state and her access to food during pregnancy: there is no unifying nutritional advice that is appro- priate for all pregnant women throughout the world, or even within nations. The well-nourished mother—does not need to ‘eat for two’ during pregnancy. Gestational metabolic adaptations allow a pregnant woman to provide energy for fetal and maternal development while making few dietary changes. Supplemental folic acid (400 µg–1 mg/ day) from peri-conception until the end of the first trimester re- duces the risk of neural tube and congenital heart defects. No other extra vitamins or micronutrients are necessary for healthy pregnant women who eat a balanced diet. Excessive vitamins and micronu- trients can be harmful to mother and fetus. The undernourished mother—needs a balanced diet that is sup- plemented with (1) vitamins, including folic acid and vitamin D, and (2) micronutrients, including iron and zinc to ensure that the fetus fulfils its growth potential. Protein and energy supplements to undernour- ished women of small stature and pelvis size improve fetal growth, but can lead to obstructed labour, a significant cause of maternal and perinatal death in low-income and middle-income countries. Around the world, obesity and excessive gestational weight gain are now more common risk factors for pregnancy complications than undernutrition. Compared with mothers who have a normal body mass index (18.5–24.9 kg/m2), obese mothers have an in- creased risk of gestational diabetes, pre-eclampsia, caesarean de- livery, stillbirth, congenital malformations and large for gestational age babies. Underweight mothers have an increased risk of small for gestational age babies, and preterm birth. Paternal obesity has a negative effect on fetal growth, probably mediated by inheritance of insulin resistance. Both low birth weight and high birth weight babies have an increased risk of obesity in later life. Introduction There is no unifying nutritional advice that is appropriate for all pregnant women throughout the world, or even within nations. Nutritional advice must take account of maternal body size, lifestyle, and availability of food. In high resource nations where food is gen- erally plentiful and obesity prevalent, dietary recommendations for a healthy pregnancy are targeted to reduce excessive weight gain and associated pregnancy complications. By contrast, in nations where food is scarce, undernourishment compromises maternal adaptation to pregnancy and leads to suboptimal fetal growth and development.

Micronutrients and vitamin supplements are given with good ef- fect to chronically undernourished pregnant women, but with few exceptions are unnecessary and can be harmful to well-nourished pregnant women. The ability of pregnant women to adapt to different environ- mental and nutritional conditions is a key requirement for repro- ductive success. Gestational metabolic adaptations minimize extra nutritional requirements and optimize fetal growth. However, millions of pregnant women are unable to provide enough nu- trition for their fetus to thrive despite these adaptations. Poor prenatal nutrition not only affects perinatal outcome, but also dictates susceptibility to adult diseases and even the health of the next generation. Maternal overweight and obesity Since the early 1970s in the United States, the prevalence of obesity (BMI >30 kg/m2) among all women aged 20–39 years has increased from less than 10% to more than 35% and is par- ticularly prevalent among non-Hispanic black women, affecting more than 55%. A similar picture is seen in the United Kingdom and to varying degrees across Europe (Fig. 14.2.1). Even in low- income and middle-income countries (LMICs), overweight/ obesity is now more prevalent than underweight. In the Americas and Caribbean, Oceania, and Europe, between 50% and 75% of all women of reproductive age are either overweight (BMI >25 kg/m2) or obese. Maternal obesity is associated with an increased risk of gestational diabetes and pre-eclampsia. During labour obese women are at in- creased risk of maternal death, haemorrhage, infection, and cae- sarean delivery. Their offspring are at greater risk of macrosomia, birth trauma, neonatal, and infant death compared with women of normal body mass index. 14.2 Nutrition in pregnancy David J. Williams

14.2 Nutrition in pregnancy 2569 Postpartum, obese mothers have greater difficulty with breast-feeding and are predisposed to postpartum weight retention. Women who had gestational diabetes are predisposed to future type-2 diabetes, and those who had pre-eclampsia are predisposed to future cardiovascular disease. The children of overweight and obese mothers are themselves pre-disposed to obesity in later life. It is therefore important for mothers to understand nutritional energy requirements before and during pregnancy to diminish the risk of transmitting obesity between generations. Maternal undernutrition Over the last 20 years in LMICs, maternal undernutrition has de- clined, such that now approximately 10% of women have a BMI less than 18.5 kg/m2 in Asia and Africa. Maternal undernutrition be- fore and during pregnancy contributes to fetal growth restriction. Low birth weight babies are at risk of perinatal and early childhood morbidity and mortality, with longer-term stunting of growth and development. Maternal and child undernutrition is the underlying cause of 3.5 million deaths around the world each year, mainly of chil- dren. Ninety per cent of the world’s undernourished children live in just 34 countries, mainly in sub-Saharan Africa, and south- central and Southeastern Asia (Fig. 14.2.2). Chronically under-nourished women are often of short stature, which increases their risk of an operative delivery due to cephalopelvic disproportion, and perioperative comorbidity is high even if caesarean section is available. In these low-income countries, 16% of babies are of low birth weight (<2500 g), of which more than two-thirds are due to fetal growth restriction and less than one-third are due to prematurity. In comparison, in high-income nations only 5% of babies are of low birth weight, of which most (55%) are premature. Low birth weight increases an individual’s risk of morbidity and mortality during all phases of life: neonatal, childhood, and adult. Weight gain in pregnancy During pregnancy, well-nourished mothers with free access to food gain up to 30% of their pre-pregnancy weight, of which only 25% is fetal. By contrast, mothers with limited access to food gain as little as 10% of their pre-pregnancy weight, of which up to 60% is fetal. Excessive and insufficient gestational weight gain is associated with an increased risk of adverse pregnancy outcome. In 2009, the National Academy of Medicine in the United States refined earlier guidelines for weight

gain in pregnancy, adjusted according to pre-pregnancy maternal weight (Table 14.2.1).
A subsequent review and meta-analysis of 1.3 million pregnant women found that almost 50% had gestational weight gain greater than these recommendations, and 23% had less. Gestational weight gain above recommendations is associated with an increased risk of large for gestational age and macrosomic offspring, preterm birth, caesarean section, gestational diabetes mellitus, pre-eclampsia, United Kingdom* Ireland* Spain* Hungary* Portugal* Austria* Greece* Italy* Germany BE: Wallonia Malta Denmark France Slovenia Poland 0.0 5.0 10.0 15.0 20.0 25.0 30.0 BE: Brussels Switzerland* Finland Norway BE: Flanders Sweden Netherlands* UK: Scotland Fig. 14.2.1 Distribution of maternal obesity (percentage of women delivering live or stillbirths with pre-pregnancy BMI ≥ 30 kg/m2) from the Euro-Peristat database and the World Health Organization (WHO). Reprinted from Devlieger R et al. (2016) Maternal obesity in Europe: where do we stand and how to move forward? A scientific paper commissioned by the European Board and College of Obstetrics and Gynaecology (EBCOG). European Journal of Obstetrics & Gynecology and Reproductive Biology, 201: 203–208. Copyright © 2016, with permission from Elsevier. *From WHO database (2009) (globally higher rates due to general female population aged 20 or older). Source: http://www.europeristat.com/our-indicators/euro-peristat-perinatal-health-indicators-2010.html.

Section 14 Medical disorders in pregnancy 2570 Fig. 14.2.2 Thirty-four countries that account for 90% of the global burden of malnutrition and 90% of children with stunted growth. Reprinted from Bhutta ZA et al. Lancet, 2013; 382: 452–77. Copyright © 2013, with permission from Elsevier.

14.2 Nutrition in pregnancy 2571 and postpartum weight retention. Women who retain weight after their first pregnancy have an increased risk of all these complications in a subsequent pregnancy. Furthermore, obesity during pregnancy fuels obesity in the next generation: about one- third of infants of obese mothers are in the ninetieth centile for their age, and a child of an overweight mother is three times more likely to be overweight by age seven years. Gestational weight gain below the National Academy of Medicine recommenda- tions increases the risk of small for gestational age offspring and preterm birth. Energy requirements during pregnancy The rate of human fetal growth is slow, and the daily incremental en- ergy stress of human pregnancy is relatively low compared with that in other species (Fig. 14.2.3). This allows a mother time to adapt her metabolism and energy expenditure to diverse nutritional condi- tions and the fetus to develop a complex brain. Energy requirements increase gradually as pregnancy progresses. In the first trimester an estimated 375 kJ (90 kcal)/day is required, in the second trimester 1200 kJ (287 kcal)/day, and in the third 1950 kJ (466 kcal)/day. The total energy cost of pregnancy for a woman with a mean weight gain of 12 kg is 325 MJ (77 000 kcal). Energy requirements during lacta- tion are similar to those of the third trimester. The three major components of energy expenditure in an average well-nourished pregnant woman are growth of the fetus and re-productive tissues (c.18%), new maternal fat stores (c.38%), and increased maternal metabolism (c.44%). Poorly nourished women try to maintain fetal growth by depressing their basal metabolic rate until late pregnancy and by laying down less fat. Although such adaptations usually result in successful reproduction, they are inev- itably a compromise with regards to perinatal health. However, at- tempts to quantify minimal energy requirements for good perinatal health will always be confounded by huge individual variability and the practical difficulties of attributing a single nutritional compo- nent to morbidity, which is a multifaceted problem. Well-nourished women increase their basal metabolic rate most rapidly after 16 weeks' gestation until term. During the

middle trimester large amounts of maternal fat are laid down as energy stores, although women with the highest cumulative rise in basal metabolic rate lay down the least fat. If food intake becomes limited during late pregnancy, maternal fat can be mobilized to support the period of most rapid fetal growth. A similar strategy of fat storage before anticipated energy expenditure is used by birds before they migrate and by mammals before they hibernate. Well- nourished women with free access to food rarely need to utilize all their fat stores to support late fetal growth, and excess fat remains difficult to lose postpartum. Even poorly nourished women with low gestational weight gain lay down some extra fat, but they also suppress their basal metabolic rate until late in pregnancy in order to support fetal growth. Maternal energy intake during pregnancy actually increases by little more than 25% of that required to fulfil energy needs. It is likely that some of the estimated shortfall is due to under-reporting 600 500 400 300 Energy cost (MJ) 200 100 0 Sweden England Netherlands Netherlands Scotland India, middle and upper class women Thailand Philippines The Gambia The Gambia, supplemented The Gambia, unsupplemented −100 Conceptus Fat deposition Maintenance Fig. 14.2.3 Estimated total energy costs of pregnancy in different nutritional environments. The ‘supplemented’ women from the Gambia were given a balanced protein–energy diet. Reproduced from Prentice AM, Goldberg GR (2000). Energy adaptations in human pregnancy: limits and long- term consequences. Am J Clin Nutr, 71 Suppl, 1226–32S, with permission from Oxford University Press. Table 14.2.1 Guidelines for gestational weight gain (recommendations of the National Academy of Medicine,
United States, 2009) Maternal
pre-pregnancy
BMI (kg/m2) Recommended weight gain at term (kg) Rate of weight gain in
second and third trimester, mean (range) kg/wk Underweight (<18.5) 12.5–18 0.51 (0.44–0.58) Normal weight (18.5–24.9) 11.5–16 0.42 (0.35–0.50) Overweight (25.0–29.9) 7–11 0.28 (0.23–0.33) Obese (>30.0) 5–9 0.22 (0.17–0.27) BMI, body mass index.

Section 14 Medical disorders in pregnancy 2572 of nutritional intake and some due to an economy of energy ex- penditure, including reduced physical activity and diet-induced thermogenesis. Metabolic changes in pregnancy Carbohydrate and fat metabolism during pregnancy During the first half of pregnancy women produce more insulin in response to a glucose load and are more sensitive to exogenous in- sulin than in the nonpregnant state. These changes affect carbohydrate and lipid metabolism to favour increased fat production and storage. During the second half of pregnancy a woman becomes resistant to insulin, so that at term the action of a particular circulating concen- tration of insulin is up to 70% lower than in the nongravid state. As a consequence, the fat stores laid down in the first half of pregnancy are mobilized and postprandial blood glucose levels remain higher for longer. Circulating levels of fatty acids and glycerol increase and are used in the maternal liver to generate energy in preference to glucose and amino acids, which are left for the fetus and placenta. Therefore, fasting pregnant women produce ketones from fatty acid oxidation far earlier than they do when not pregnant. Women with a defect in fatty acid oxidation and who fast during pregnancy have limited ketogenesis and are vulnerable to multiorgan energy failure, including ‘acute fatty liver’ and hypoglycaemia. Other pregnant women with an exaggerated peripheral resistance to insulin are at risk of gestational diabetes mellitus. Protein metabolism Pregnancy is an anabolic state. Protein and nitrogen metabolism adapt early and gradually throughout healthy pregnancy to provide for tissue growth. Well-nourished women are estimated to accumu- late an extra 500 g to 1 kg of protein during pregnancy, almost half of which is maternal lean body mass, while the rest lies within the fetus and reproductive tissues. In

the United Kingdom the advised increment of dietary protein has been calculated to increase gradually throughout pregnancy to 8.5 g/day at term, but this does not consider reduced hepatic metab- olism of branched chain amino acids and hence reduced urea syn- thesis. The rate of urea synthesis declines by 30% during the first trimester and by 45% during the third trimester, providing more nitrogen for protein synthesis. Consequently, maternal serum urea concentration continues to fall in the third trimester, despite a fall in glomerular filtration rate that raises the serum creatinine levels. Vitamins and micronutrients In many parts of the developing world, micronutrient deficiencies are endemic and have serious consequences for fetal, neonatal, and maternal well-being (e.g. hypothyroidism due to iodine deficiency and night blindness due to vitamin A deficiency). Such deficiencies are rare in developed countries. Calculated increments in the recommended daily allowance of specific nutrients are derived from estimates of the cost of fetal growth and increased maternal metabolism. However, these calcula- tions do not usually take account of maternal metabolic adaptations aimed to minimize the need for extra nutrients. For example, intes- tinal absorption of calcium increases in well-nourished women and the need for an increase in dietary calcium diminishes. Conversely, increased folic acid excretion leads to an underestimate of folic acid requirements. Furthermore, individual micronutrients interact with each other and changes to one may have a detrimental effect on the activity of another. Folic acid Supplemental folic acid (400 µg to 1.0 mg/day) or folic acid food for- tification during the first trimester reduces the risk of fetal neural tube defects (NTD) and congenital heart defects (CHD). Women who have previously had a pregnancy affected by NTD or CHD, or who smoke, are obese, diabetic, or who take antifolate medica- tion should take a higher dose of folic acid, up to 5 mg folic acid daily. With this exception, extra vitamins and micronutrients are not necessary for well-nourished, healthy pregnant women who eat a balanced diet. Indeed, excessive amounts of certain micronutrients can be harmful to the fetus. The situation is quite different for under- nourished women in LMICs. Iron During pregnancy, expansion in plasma volume exceeds the in- crease in red cell mass, causing a fall in haemoglobin concentration. Healthy pregnant women not taking iron supplements drop their haemoglobin from 133 g/litre to 110 g/litre by 36 weeks’ gestation. The minimum incidence of low birth weight (<2500 g at term) and preterm labour is associated with maternal haemoglobin in the range 95–105 g/litre, which in the nongravid state would indicate an-aemia. A meta-analysis of randomized controlled trials examining the benefit of supplemental iron found a significant reduction in the proportion of women with haemoglobin levels less than 100 g/litre, but no effect—beneficial or harmful—on maternal or fetal outcome. In otherwise well-nourished women in the United Kingdom, rou- tine supplemental iron is not recommended. Anaemia of multiple causes is endemic in many LMICs, and the risk of maternal death is increased with severe anaemia (haemo- globin <70 g/litre), a condition where supplemental iron is unlikely to have much effect. However, in such countries mild to moderate anaemia can be prevented with iron (60 mg daily) and folate sup- plementation, which improve birth weight without increasing the maternal risk of Plasmodium infection. Many LMICs therefore ad- vocate a policy of iron and folic acid supplementation for all preg- nant women. More studies are necessary to monitor the effects of this policy on maternal and perinatal outcome. Anaemia in preg- nancy is discussed in more detail in Chapter 14.17. Vitamin A Vitamin A is a lipid-soluble vitamin essential for healthy embryogen- esis and fetal growth. Preformed vitamin A is found in dairy prod- ucts and liver. Vitamin A deficiency is endemic in some parts of the world and associated with night blindness. In these circumstances, maternal vitamin A supplements may result in a small increase in birth weight. However, excessive doses of vitamin A (>15 000 IU/day or 5000 mcg/day) are teratogenic, and drugs that are derived from vitamin A, such as the retinoids, are associated with an estimated

25-fold increased risk of fetal malformation. The National Institute for Health and Care Excellence (NICE) in the United Kingdom re- commends up to 700 mcg vitamin A daily to preserve health and re- duce the risk of teratogenesis. In general, vitamin A supplements are

14.2 Nutrition in pregnancy 2573 unnecessary for well-nourished women, potentially harmful to the fetus, and of unproven benefit to safely improve fetal growth. Breast milk is rich in vitamin A and is important for neonatal immunity. Thiamine (vitamin B1) Thiamine deficiency is endemic in some developing countries but is also a global problem in women with hyperemesis gravidarum. Severe and persistent vomiting during pregnancy leads to thiamine deficiency and rarely causes Wernicke's encephalopathy, hence thia- mine replacement is recommended for women with this condition. Vitamins C and E Serum vitamin C levels fall by about 50% during pregnancy, hence it was previously recommended that this was supplemented. Furthermore, the antioxidant properties of vitamins C and E were thought to reduce the risk of pre-eclampsia, but this is not the case. Indeed, at high doses (vitamin C 1 g/day and vitamin E 400 IU/day) these vitamins increase the risk of babies being born with a low birth weight and their supplementation is not justified in pregnancy. Calcium and vitamin D The growing fetus gains about 50 mg calcium per day by mid preg- nancy and about 300 mg/day at term, and the breastfed infant receives about 250 mg of calcium in breast milk each day. The re- commended daily allowance of calcium during pregnancy and lacta- tion is 1.2 g/day, but women with much less dietary calcium undergo metabolic adaptations to meet the demands of pregnancy and lacta- tion without any detriment to their health or that of the fetus. During pregnancy, maternal calcium absorption increases two- fold, stimulated by increased 1,25-dihydroxyvitamin D (calcitriol) activity due to placental synthesis and increased renal 1α- hydroxylase activity. Although urinary calcium excretion doubles during pregnancy, fasting urinary calcium excretion, corrected for increased creatinine clearance is unchanged. The concentration of parathyroid hormone falls during pregnancy, suggesting that the pregnant woman receives enough calcium for her growing fetus. There are two caveats: one is the pregnant adolescent who needs to meet the demands of her own bone growth as well as that of the fetus; the other is the benefit of supplemental calcium for women on a low-calcium diet to prevent pre-eclampsia. Following delivery, circulating calcitriol concentrations return to nonpregnant levels. During the first six months of breastfeeding mineralization of the maternal axial skeleton declines by about 3– 5%, recovering after six months whether or not breastfeeding con- tinues. Calcium supplements of 1 g/day given to lactating women do not prevent bone demineralization or improve the calcium concen- tration of breast milk, even if the woman is on a low-calcium diet. Furthermore, repeated long periods of breastfeeding in women with a low calcium intake do not contribute to osteoporosis in later life. Vitamin D deficiency is prevalent around the world, but is par- ticularly common among women who are obese, dark-skinned, avoid sun exposure, and who have a sedentary lifestyle. However, there is little consensus among international organizations on the recommended plasma level of 25(OH)vitamin D levels for a healthy pregnancy outcome, these ranging between over 30 to more than 75 nmol/litre. Similarly, the recommended dose of vitamin D sup- plementation for women with vitamin D deficiency ranges between 200 to 2000 IU daily. These wide ranges reflect a lack of good quality clinical trial data. We still need to define harmful vitamin D defi- ciency in pregnancy and what dose and dose-frequency of vitamin D supplementation (if any) can safely and effectively correct it. Polyunsaturated fatty acids Maternal consumption of supplements containing n–3 long- chain polyunsaturated fatty acid may reduce the risk of preterm birth (<34 weeks) and childhood allergy but have no proven benefit on neurocognitive and retinal development in the fetus.

Supplementation with more than 200 mg of docosahexaenoic acid per day, which can be achieved with the consumption of one or two portions of sea fish per week, may protect against preterm birth. Limiting fish intake to smaller oily fish such as herring, mackerel, and salmon will diminish the overstated concern that large predator fish contaminated by neurotoxic levels of methylmercury might be harmful to the fetus. Iodine Almost two billion people have at least mild iodine deficiency from living in iodine-deficient areas. Inadequate dietary iodine leads to maternal hypothyroidism, which in turn is detrimental to fetal growth and development. Supplemental iodine, which is added to salt in most developed countries, can prevent these consequences. Zinc Zinc deficiency is associated with intrauterine growth restriction and teratogenesis. Maternal zinc levels remain stable during preg- nancy through increased intestinal absorption. Excess iron supple- ments, smoking, alcohol abuse, or subsistence cereal diets high in phytate can all inhibit zinc absorption. Under such conditions, preg- nant women may benefit from 25 mg zinc daily. Other dietary matters Foods to avoid during pregnancy Acute maternal infection with Toxoplasma gondii can cross the pla- centa to the fetus. Congenital infection is least likely during early pregnancy, but more severe when it occurs. The risk of congenital in- fection can be kept to a minimum by not eating undercooked meat, taking care while handling raw meat, and avoiding contact with cat faeces. The risk of Listeriosis infection can be reduced by eating and drinking only pasteurized dairy products. The risk of salmonella in- fection can be reduced by avoiding uncooked eggs, or meat. Food cravings during pregnancy Common food cravings during pregnancy are for dairy products and occasionally for nonorganic material such as soil (pica). Common aversions are to alcohol, caffeine, and meats. Fetal programming—the influence of fetal nutrition on adult disease Epidemiological studies have found that low birth weight due to fetal growth restriction, rather than prematurity, is associated with an increased risk of cardiovascular disease in adulthood. It is

Section 14 Medical disorders in pregnancy 2574 hypothesized that a poorly growing fetus makes metabolic adapta- tions in utero to optimize growth and development. Despite these physiological adaptations, driven in part by insulin-like growth fac- tors, birth weight remains low, and because of them the individual is programmed to insulin-resistance syndromes including future car- diovascular disease. Animal studies have shown that the composition of maternal diet can influence fetal growth and consequently blood pressure in her offspring. At present not enough is known about the mechan- isms that control human fetal growth to give a mother nutritional advice that might eventually reduce the risk of cardiovascular dis- ease in her children. Inheritance of insulin resistance from father to fetus tempers insulin-mediated fetal growth and may be another explanation for the association between low birth weight and future type-2 diabetes/metabolic syndrome. Understanding these mech- anisms may help to ameliorate the global epidemic of cardiovas- cular disease. FURTHER READING Bhutta ZA, et al. (2013). Evidence-based interventions for improve- ment of maternal and child nutrition: what can be done and at what cost? Lancet, 382, 452–77. Black RE, et al. (2013). Maternal and child undernutrition and over- weight in low-income and middle-income countries. Lancet, 382, 427–51. Chitayat D, et al. (2016). Folic acid supplementation for pregnant women and those planning pregnancy: 2015 update. J Clin Pharm, 56, 170–5. Devlieger R, et al. (2016). Maternal obesity in Europe: where do we stand and how to move forward?: a scientific paper commissioned by the European Board and College of Obstetrics and Gynaecology (EBCOG). Eur J Obstet Gynaecol Reprod Biol, 201, 203–8. Goldstein RF et al. (2017). Association of gestational weight gain with maternal and infant outcomes: a systematic review and meta- analysis. JAMA, 317, 2207–25. Koletzko B, et al. (2014). Current information and Asian perspectives on long-chain polyunsaturated fatty acids in pregnancy, lactation and

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