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INFLUENCE OF EXERCISE ON PRECONCEPTION, PREGNANT WOMEN, THE DEVELOPING FOETUS AND DELIVERY: A REVIEW

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ABSTRACT

There are many questions about the relationship between pregnancy and exercise. This article presents an overview of published works on a) exercise and problems with conception, with the focus on amenorrhea and miscarriage, b) exercise and pregnancy, highlighting the influence of exercise on maternal metabolism, cardiovascular and respiratory function, c) exercise and the developing embryo/foetus, with the focus on placental formation/growth, substrate availability and foetal hyperthermia, d) exercise and the labour/delivery process, with the emphasis on preterm labour, as well as e) exercise and the postpartum period, with the focus on neonatal outcome, maternal recovery after parturition and lactation. It concludes with recent recommendations and precautions associated with exercise during pregnancy, as well as shortcomings in available information. In general, literature does not seem to support the concerns about prenatal exercise, but rather shows that both mother and foetus benefit when the mother is involved in moderate exercises during pregnancy. Indications are that the benefits will only be experienced in full by women who, within reasonable limits, continue to exercise throughout their pregnancy.

Key words: Exercise, reproduction, pregnancy, foetal, maternal care.

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INTRODUCTION

Exercise has become a vital part of many women's lives. Although many women still prefer to engage in exercise during pregnancy they are concerned about the possible adverse effects. The concerns about exercising during pregnancy relate to the dual stresses of pregnancy and exercise that might create conflicting physiological demands on the mother-to-be (Wiswell, 1996; Sternfeld, 1997). In contrast to the adaptations that occur during pregnancy, which are intended to nurture and protect the foetus, those that occur during exercise serve to maintain maternal homeostasis. Excessive physical activity during pregnancy may thus create conflicting maternal and foetal needs and may pose potential risks for the outcome of the pregnancy. On the other hand, the adaptations may complement each other and offer potential benefits (Sternfeld, 1997).

The physiological changes that occur during exercise include: redistribution of cardiac output away from the visceral circulation to the exercising muscles and skin, depletion of energy stores and an increase in body temperature (Clapp, 1996). In addition, several other potential risks have been described, including teratogenic effects as a result of exercise-induced hyperthermia, decreased carbohydrate availability for the foetus, redistribution of uterine blood flow with subsequent foetal hypoxia, increased

uterine contractility with a possible increase in the risk for preterm labour, infertility, abortion, congenital malformation, cord entanglement, placental separation, premature membrane rupture, growth restriction, foetal trauma, foetal bradycardia, difficult labour, as well as maternal musculoskeletal injury (Clapp, 1996; Sternfeld, 1997; Stevenson, 1997; Clapp, 2000; Borg-Stein, Dugan & Gruber, 2005).

Exercise recommendations during pregnancy have evolved over the last several decades (Borg-Stein *et al*., 2005). Until the 20th century physical activity during pregnancy had been discouraged primarily because of theoretical concerns of exercise-induced injury and adverse foetal and maternal outcomes (Dempsey, Butler & Williams, 2005). Consequently many saw pregnancy as a state of confinement in which women were not encouraged to engage in recreational physical activity (Dempsey *et al*., 2005). Results from animal studies published before the 1970's clearly supported these concerns, which led to a variety of restrictive regulations regarding exercise (Clapp, 1996; Dempsey *et al*., 2005). Women were instructed to limit their involvement in exercise and non-exercisers were told not to initiate exercise when pregnant (Borg-Stein *et al*., 2005). In contrast, research findings published since the 1970's do not support these concerns (Clapp, 2000; Dempsey *et al*., 2005). In fact, findings of studies completed since 1985 have demonstrated no adverse maternal or foetal effects in healthy women engaged in mild and moderate exercise activities (Clapp, 1996; Henriksson-Larsen, 1999; Riemann & Kanstrup-Hansen, 2000; Dempsey *et al*., 2005;), but rather, showed somewhat favourable effects (Wiswell, 1996; Sternfeld, 1997; Clapp, 2000; Frey, 2002). The influence of the past 30 years of research can clearly be seen in the significant changes of the American College of Obstetricians and Gynaecologist's (ACOG) guidelines, published in 2003 (Committee on patient education of the ACOG, 2003). These guidelines recommend moderate exercise, 30 minutes or more per day, on most, if not all days of the week for women with low risk pregnancies.

For the first time, official recommendations advocated exercise for previously sedentary pregnant women and those with medical or obstetric complications, although only after they have undergone medical evaluation and clearance. Pregnancy is thus no longer regarded as a medical condition requiring the endorsement of a sedentary lifestyle (Dempsey *et al*., 2005; Morris & Johnson, 2005), but as a normal physiological condition.

EXERCISE AND PROBLEMS WITH CONCEPTION

A high incidence of reproductive disorders is frequently reported for female athletes who engage in strenuous training programmes, of which amenorrhea (primary and secondary), oligomenorrhea and luteal phase deficiencies are well known (Thong & Graham, 1999; Greydanus & Patel, 2002). Proposed causative factors for such exercise-associated reproductive disorders include negative energy balance, the psychological and physical stress of exercise, extreme leanness (inadequate body fat), nutritional deficiencies and exacerbation of any genetic predisposition. It goes without saying that the occurrence of these disorders is dependent on the intensity and duration of exercise training, the type of exercise, age, and other factors (Thong & Graham, 1999; Greydanus & Patel, 2002).

Amenorrhea and ovulatory disturbances

The most commonly reported disorder is secondary amenorrhea, or cessation of menstrual function (Thong & Graham, 1999). The amenorrhea noted in athletes is often referred to as a hypothalamic amenorrhea, associated with abnormal gonadotropin releasing hormone (GnRH) and luteinising hormone (LH) (Greydanus & Patel, 2002). The prevalence of secondary amenorrhea is estimated to be as high as 44% among female athletes (Thong & Graham, 1999) and up to 66% in elite athletes (Greydanus & Patel, 2002), compared to 1-5% occurrence in the general population (Thong & Graham, 1999). Menstrual difficulties have been reported in 5-20% of vigorously exercising females, 12% of swimmers and cyclists, 44% of ballet dancers, 50% of tri-athletes and 51% of endurance runners (Greydanus & Patel, 2002).

It is well known that rapidly progressive increases in an individual's exercise regimen, or very intense competitive training, can suppress the hypothalamic-pituitary-ovarian axis with resultant ovulatory dysfunction (Clapp, 1996). Most of the studies concerning exercise-induced amenorrhoea have focused on low body weight and low fat ratio to body weight (Thong & Graham, 1999); (Sundgot-Borgen, 2000). Previous theories that menses could not take place below 17% body fat have not been substantiated (Greydanus & Patel, 2002). Recent findings suggest that low energy availability/nutrient deficiency and energy drain, rather than inadequate body fat and exercise stress are the more likely mechanisms by which exercise impinges negatively on the hypothalamic-pituitary-ovarian axis in female athletes (Thong & Graham, 1999; Sundgot-Borgen, 2000).

Reproductive function is invariably related to whole-body energy balance. The dependence of reproduction on energy availability led to the proposal that reproductive disturbances in female athletes may not be related to exercise per se, but rather to inadequate energy intake in order to meet the increased demands of exercise. It is known that the gonadotropin releasing hormone (GnRH) pulse generator is disrupted by acute and chronic negative energy balance in humans. Subsequently gonadotropin secretion from the pituitary is altered and ovarian function is suppressed. The low-energy hypothesis is supported by observations that amenorrheic athletes often display signs of energy insufficiency, including low triiodothyronine (T3) levels (which are most likely responsible for the decrease in resting metabolic rate), mildly elevated cortisol levels and altered insulin and glucose levels. These changes, along with suppressed reproductive function are probably elicited as adaptive responses to conserve metabolic fuels (Thong & Graham, 1999).

Leptin has been proposed as a factor in regulating reproduction and has a potential link to exercise-associated reproductive disorders in female athletes. This adipocyte-derived hormone has recently been proposed to play an integral part in the interaction between nutrition and reproduction.

Leptin, in addition to regulating body-weight homeostasis and preventing obesity, signals a depletion of energy stores with subsequent initiation of appropriate physiological responses. It has been showed that when leptin falls below a critical level, due to energy deficiency (as experienced in female athletes), it coordinates the compensatory responses, including suppression of reproduction and thyroid-induced thermogenesis. Thus leptin integrates adipose tissue stores, energy homeostasis and the hypothalamic regulators of reproduction and as such it provides a critical link between fat and fertility (Thong & Graham, 1999). It is thus important that physically active women should be aware of the importance of sufficient energy intake to sustain their regular menstrual cycle (Sundgot-Borgen, 2000).

Another factor implicated, in the preconception problems of the recreationally active female, is the induction of subtle ovulatory disturbances, such as luteal phase abnormalities, during prolonged, but moderate exercise training. The cycles of such athletes appear to have a prolonged follicular phase that compensates for a shortened luteal phase, resulting in a cycle with normal duration. Since the presence of regular menstrual bleeding is often seen as an indication that the hypothalamic-pituitary-ovarian axis (HPO axis) functions normally, these less conspicuous reproductive disturbances are often undiagnosed. Thus, it can not be assumed that all physically active "regularly menstruating" women have normal reproductive function (Thong & Graham, 1999). Abnormal follicular development, suppressed ovarian function in both amenorrheic and eumenorrheic athletes, as well as decreased progesterone levels in the luteal phase, have been reported in recreationally active women. The primary cause has been identified to be at the level of the hypothalamus, rather than decreased pituitary responsiveness to gonadotropin releasing hormone (GnRH) (Thong & Graham, 1999).

Spontaneous abortion/ miscarriage

Young women, who are training on a regular basis, may for several weeks not recognize that they are pregnant such that

pregnancy could be confused with a training-induced amenorrhoea. In view of the susceptibility of the foetus to injury in the early stages of pregnancy it is important that all training programmes for fertile women be compatible with a safe pregnancy (Shephard, 2000; Magann, Evans, Weithz & Newnham, 2002). Currently many studies indicate that the incidence of spontaneous abortion/miscarriages and ectopic pregnancies are the same for women who continue to exercise moderately during pregnancy and for control groups (Clapp, 1996; Sternfeld, 1997; Clapp, 2000; Riemann & Kanstrup-Hansen, 2000; Magann *et al*., 2002; Morris & Johnson, 2005). However, Hjollund *et al*. (2000) found an increased risk of spontaneous abortion among women who engage in high intensity physical activity during the time of implantation of the embryo. It would appear that these abortions typically occurred 2 weeks after implantation, with no significant effect in other time windows during early pregnancy. Nevertheless, it would be desirable to see further research clarification on this issue.

EXERCISE AND PREGNANCY

Psychological effects

Various studies have found that women who continue to exercise throughout pregnancy, or who start an exercise programme during pregnancy, derive mental benefits including: a decrease in perceived stress (Clapp, 2000), lower incidence of depression (Henriksson-Larsen, 1999; Clapp, 2000) greater sense of well-being (Clapp, 1996; Margolis, 1996; Sternfeld, 1997; Wang & Apgar, 1998; Clapp, 2000; Morris & Johnson, 2005), improved body-image (Sternfeld, 1997; Clapp, 2000), improved attitude (Clapp, 2000) and an enhanced self-image (Clapp, 1996; Sternfeld, 1997; Henriksson-Larsen, 1999; Morris & Johnson, 2005). The biological mechanisms underlying these positive effects probably include hormonal and metabolic adaptations associated with improved cardiovascular functioning, alterations in catecholamine release and response, and increases in endogenous opiates above that which occurs with pregnancy itself (Sternfeld, 1997). However, two weeks after cessation of exercise these positive effects apparently

start to diminish (Morris & Johnson, 2005).

Discomforts/symptoms of pregnancy

Pregnancy is usually associated with various discomforts. Several studies indicate that women who exercise during pregnancy, especially in the last trimester, have a lower incidence, than sedentary groups, of symptoms, such as nausea, fatigue, leg cramps, swelling, round ligament pain, backache, nausea, heartburn, low back pain, shortness of breath, somatic complaints, musculoskeletal complaints and insomnia (Clapp, 1996; Horns, Ratcliffe, Leggett, & Swanson, 1996; Sternfeld, 1997; Magann *et al*., 2002; Morris & Johnson, 2005; Lochmuller & Friese, 2005). A discussion of the underlying mechanisms is largely beyond this article.

Maternal weight gain

It is still controversial whether moderate exercise has a significant influence on weight gain in the pregnant woman. While the majority of studies points towards a lower gain in weight (Clapp, 1996; Clapp, 2000; Magann *et al*., 2002; Lochmuller & Friese, 2005) and fat deposits, not all studies could confirm these.

If the various publications on this subject are considered it would appear that the effect of exercise on maternal weight is dependent on the amount and intensity of exercise and the stage of pregnancy and that the difference between moderately exercising and non-exercising women, are really only significant during the last trimester when exercising women show a smaller gain in weight (Hale & Milne, 1996; Clapp, 2000; Magann *et al*., 2002; Morris & Johnson, 2005). This is perhaps only relevant to women who are aware of the influence of their life-styles on their pregnancies, because in most studies the women volunteers chose to participate in the exercise groups. It is possible that these women volunteers had desirable eating habits and healthier life-styles, thus leading to less weight gain regardless of exercise status (Morris & Johnson, 2005).

Respiratory function

In general pregnant women experience elevation of the diaphragm by the developing foetus. This reduces residual lung volume, expiratory reserve volume

and total lung volume. The resting respiratory minute volume would appear to be increased and the dyspnoea threshold reduced (Shephard, 2000). In the non-pregnant state, regular aerobic exercise produces a set of physiological adaptations, including increased maximal oxygen consumption (VO_2 max) and decreased sub-maximal heart rate, which result in higher aerobic capacity and improved exercise performance. The combination of regular exercise and pregnancy may help to maintain VO_2max (Hale & Milne, 1996; Sternfeld, 1997; Wolfe & Weissgerber, 2003) or even to improve it, particularly among previously sedentary women and women who continue to exercise during pregnancy (Hale & Milne, 1996; Sternfeld, 1997; Wolfe & Weissgerber, 2003). Collins (1983) reported that women who started a stationary cycling exercise program in the third trimester and who continued with it, had an 18% improvement in their absolute aerobic capacity (liters of oxygen per minute), while the non-exercising control group had a 4% decline in absolute aerobic capacity. Controlled prospective studies support these findings (Wolfe & Weissgerber, 2003). However, there are still contradictions about the effects of exercise on ventilation threshold, peak respiratory gas exchange ratio (Shephard, 2000), pulmonary reserve, alveolar ventilation and tidal volume (Hale & Milne, 1996). It is important to note that during the late stages of pregnancy, the increase in body size and mass, plus a restriction of abdominal breathing, make vigorous exercise quite difficult (Shephard, 2000).

Metabolism

During pregnancy a changed glycaemia response to exercise is seen (Riemann & Kanstrup-Hansen, 2000). The typical exercise-induced initial hyperglycaemia of non-pregnant subjects appears to be reversed in healthy pregnant women who regularly engage in recreational physical activities (Dempsey *et al*., 2005). It was shown that from the 8th gestational week the initial increase in blood glucose levels was only seen at exercise intensities greater than 80% of maximum, and that, by the 23rd week and onwards, the response is actually towards a decrease in blood glucose.

This appears to be accompanied by the absence of expected changes in insulin and catecholamine responses (Hale & Milne, 1996). It is suggested to be due to increased foeto-placental energy demands (Borg-Stein *et al*., 2005).

In theory, the exercise-induced caloric expenditure of the mother can have adverse effects on multiple aspects of the course and outcome of pregnancy (Clapp, 2000). A drop in maternal blood glucose levels during exercise could potentially reduce the foetal glucose delivery and thus influence foetal growth (Riemann & Kanstrup-Hansen, 2000; Shephard, 2000; Dempsey *et al*., 2005). Thus, it is important to ensure that a heavy and sustained bout of maternal exercise, does not induce marked maternal hypoglycaemia. Some evidence exists that, perhaps as a means of protecting the foetus from hypoglycaemia, pregnancy might perhaps reduce the ability of the mother to metabolize carbohydrates. This concurs with the previously mentioned lack of glycaemic response during exercise. If so, this could limit the ability of the mother to perform anaerobic activities during pregnancy, thereby protecting the foetus's needs (Shephard, 2000).

Cardiovascular function

The cardiovascular changes associated with pregnancy include: an increased resting heart rate (Wiswell, 1996; Sternfeld, 1997; Shephard, 2000; Wolfe & Weissgerber, 2003; Morris & Johnson, 2005), a decreased maximum heart rate (Wiswell, 1996; Hale & Milne, 1996; Sternfeld, 1997; Wolfe & Weissgerber, 2003), an increased cardiac output, an increased stroke volume, an increased left ventricular mass (Pivarnik, 1996; Shephard, 2000) and an increased blood and plasma volume (Shephard, 2000; Morris & Johnson, 2005). Most of these changes are seen as early as in the fifth week of gestation and by 12 weeks, the rise in cardiac output is about 35% above pre-pregnancy levels (Morris & Johnson, 2005). For the exercising pregnant woman who uses her heart rate as a training guide, the pregnancy-induced increased resting heart rate and decreased maximum heart rate could compromise heart-rate based predictions of aerobic fitness and training intensity (Hale & Milne, 1996; Wiswell, 1996; Sternfeld,

1997; Shephard, 2000; Wolfe & Weissgerber, 2003; Borg-Stein *et al*., 2005). The use of revised pulse rate target zones (Wolfe & Weissgerber, 2003), or the "talk test" (the mother is over exercising if unable to maintain a normal conversation) (Borg-Stein *et al*., 2005), along with perceived exertion is recommended to prescribe exercise intensity during pregnancy (Sternfeld, 1997; Wolfe & Weissgerber, 2003).

Various studies have found that the typical cardiovascular changes seen in pregnant women are amplified when she exercises (Clapp, 1996; Hale & Milne, 1996; Pivarnik, 1996; Sternfeld, 1997; Avery, Stocking, Tranmer, Davies, & Wolfe, 1999; Clapp, 2000; Shephard, 2000; Clapp, 2003; Morris & Johnson, 2005). Plasma volume is known to increase both as a result of pregnancy and as a result of exercise (Clapp, 1996, Hale & Milne, 1996; Clapp, 2000; Clapp, 2003). Pregnant women who exercise have blood volumes which are, in general, about 20% greater than their sedentary controls (Clapp, 2000; Morris & Johnson, 2005) and it can be as much as 35% in women who participate in endurance training (Hale & Milne, 1996; Sternfeld, 1997; Shephard, 2000). This increase may improve physical ability during the first few months, but the advantage may later be offset by a significant increase in body mass (Shephard, 2000). It is generally assumed that the increased blood volume is accompanied by an increase in red cell volume, haemoglobin and total red blood cell count (Hale & Milne, 1996; Sternfeld, 1997). However, anaemia as a result of dilution by a disproportional increase in plasma volume is not uncommon.

As for plasma volume, both pregnancy and exercise can increase cardiac output (Clapp, 1996; Clapp, 2000; Shephard, 2000; Clapp, 2003; Morris & Johnson, 2005). This is not merely the result of the increase in plasma volume. According to Clapp (2000), women who continue to exercise during pregnancy have a 10% greater change in end-diastolic volume and stroke volume, in comparison with non-exercisers. The changes in cardiac output seem to persist for up to one year postpartum and are accentuated by subsequent pregnancies.

Cardiac output, postpartum, was shown by Clapp (2000) to be 11% higher and the total peripheral resistance 11% lower than before pregnancy. This data suggests that the combination of exercise and pregnancy promotes vasodilation with a resultant decrease in barotraumas, which may protect against vascular disease later in life. It goes without saying that a pregnant woman's cardiac output during physical activity could be affected by gestational age, body position, exercise intensity and modality, and fitness level (Pivarnik, 1996).

Other reported exercise-induced cardiovascular changes include: increased intervillous space blood volume (Clapp, 2003), dilation of all four cardiac chambers, increased aortic diameter, enlargement of the venous capacity vessels (Shephard, 2000) and increased heart rates of 10-15 beat/min during submaximal exercise (Wiswell, 1996; Pivarnik, 1996; Avery *et al*., 1999; Shephard, 2000). In contrast mean arterial pressure response does not seem to be affected by pregnancy (Pivarnik, 1996; Avery *et al*., 1999). The latter may be related to the fact that increased blood flow to the uteroplacental unit, skin and kidneys causes a concomitant decrease in systemic vascular resistance (Pivarnik, 1996).

It was further shown that left ventricular function is maintained even during vigorous exercise in healthy pregnant subjects during the second half of pregnancy. In early pregnancy, the left ventricle adapts to strenuous bicycle exercise by increasing its contractile reserve, enhancing ventricular emptying, whereas in late pregnancy, the left ventricle increases its preload reserve without significantly increasing its contractile reserve. Women are thus "cardiovascularly" disadvantaged early in pregnancy (Veille, 1996). Using Doppler signals, -it was found that diastolic filling patterns are significantly influenced by pregnancy and that they differ depending on the trimester of pregnancy (Veille, 1996). It is important to note that in the late stages of pregnancy the foetus may shift to a position where maternal venous return is compromised, particularly if physical activity is performed while lying supine (Shephard, 2000).

EXERCISE AND THE DEVELOPING EMBRYO/FOETUS

Placenta formation/growth

Placental development is altered by a variety of environmental factors that alter placental bed blood flow and/or oxygen delivery. In a study that examined the effects of running throughout pregnancy on villous vascular development and cell proliferation, it was found that continuing to run regularly throughout pregnancy leads to an increase in both absolute and relative vascular volume and cell proliferation at term. It has been suggested that this effect may have clinical value in cases at risk for anomalous foeto-placental growth, as increased villous vascular volume should improve foeto-placental growth by enhancing placental transfer of oxygen and diffusible substrate (Bergmann, Zygmunt & Clapp, 2004). In women who continue strenuous weight-bearing types of exercises it would appear that exercise in early and mid pregnancy stimulates placental growth (Clapp, 1996; Clapp, 2000; Clapp, 2006), without increasing the frequency of placental abnormalities (Clapp, 2000). Increased villous surface area (Riemann & Kanstrup-Hansen, 2000), increased size of larger villi and increased placental volumes have also been reported as results of prenatal exercise (Hale & Milne, 1996).

Substrate availability/Glucose availability

The delivery of oxygen and other substrates to the maternal-foetal interphase is the major maternal environmental stimulus which either up- or down-regulates foeto-placental growth. During pregnancy, sustained exercise sessions cause an intermittent reduction in oxygen and substrate delivery to this interphase (Riemann & Kanstrup-Hansen, 2000; Wolfe & Weissgerber, 2003; Clapp, 2003; Clapp, 2006), which may transiently reduce foetal glucose availability and thus influence foetal growth (Riemann & Kanstrup-Hansen, 2000; Wolfe & Weissgerber, 2003). It has been suggested that regular bouts of sustained exercise may improve oxygen and substrate delivery at rest (Clapp, 2003; Clapp, 2006). However, this depends on the type of maternal carbohydrate intake (low- versus high-glycaemic sources) and food intake frequency, that in turn may influence

substrate availability through their effects on maternal blood glucose levels and insulin sensitivity (Clapp, 2006).

Cardiovascular effects

The increased maternal blood volume accompanying pregnancy serves the expanded vascular system of the uterus and the growing foetoplacental unit (Sternfeld, 1997). However, during exercise cardiac output is redistributed away from the splanchnic organs, to the skeletal muscles and skin (Sternfeld, 1997; Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005; Dempsey *et al*., 2005). This well-known response to exercise is the basis for the concern that exercise during pregnancy may result in a decreased blood flow to the uterus, placenta and foetus, which may induce foetal/placental hypoxia and may compromise foetal growth by depriving the foetus of oxygen and nutrition (Sternfeld, 1997; Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005; Dempsey *et al*., 2005). It has been suggested that compensatory mechanisms/adaptive responses may act to maintain uterine blood flow, and oxygen and substrate delivery, including: a) the marked rise in maternal haematocrit which occurs with exercise, which decreases relative plasma volume and increases the oxygen carrying capacity of the blood, b) an inverse relationship between blood flow and oxygen-extraction in which arterio-venous oxygen difference increases as flow decreases, c) redistribution of the blood flow which favours the placenta over the myometrium (Sternfeld, 1997; Shephard, 2000) and d) increased oxygen extraction (Morris & Johnson, 2005). In addition, conditioning may lessen the exercise-induced decrease in uterine blood flow (Morris & Johnson, 2005). As a result, oxygen delivery to the foetus and foetal VO_2 do not appear to be compromised during maternal exercise (Clapp, 1996; Sternfeld, 1997; Dempsey *et al*., 2005). In fact, it would appear that the intermittent reduction in uterine blood flow may have a stimulatory effect on placental growth (Clapp, 2000). An increased foetal heart rate (FHR) is found during maternal exercise (Hale & Milne, 1996; Avery *et al*., 1999; Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005), especially during high levels of exertion (Dempsey *et al*., 2005).

This increase is dependent on the training intensity and duration, gestational age, maternal level of fitness and the type of sport (Riemann & Kanstrup-Hansen, 2000). Some hypotheses about the cause of the increase in foetal heart rate include: arousal of the foetus into the awakened state, placental transfer of maternal catecholamines, a small reduction in oxygen delivery, direct stimulation of the foetus by maternal movements, mild transient stress and increases in foetal temperature (Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005). The usual foetal reaction to sustained submaximal maternal exercise would be an increase in the baseline resting foetal heart rate, with a reduction in foetal heart rate reactivity and breathing movements (Shephard, 2000; Wolfe & Weissgerber, 2003).

Moderate transient foetal bradycardia has been found immediately following a bout of maternal activity (Avery *et al*., 1999; Shephard, 2000; Wolfe & Weissgerber, 2003; Morris & Johnson, 2005). This may occur as a result of an abrupt decrease in maternal cardiac output (Morris & Johnson, 2005). In addition, occasional abnormalities of the foetal heart rate and ultrasound evidence of foetal flow redistribution have been observed during training or testing on stationary cycle ergometers (Clapp, 2000). According to Veille, (1996), exercise does not seem to influence the resistivity index of the umbilical artery, and may even cause it to decrease. In addition, studies have shown an unchanged flow in umbilical circulation during exercise (Hale & Milne, 1996; Riemann & Kanstrup-Hansen, 2000). It would further appear that the ventricular diastolic filling properties of the foetal heart are not influenced by maternal bicycle exercise (Veille, 1996).

Foetal hyperthermia

Exercise induces an increase in body temperature (Dempsey *et al*., 2005). At rest, foetal temperature is about 0.5^{o}C higher than maternal temperature. This gradient facilitates the dissipation of foetal heat by transferring it to the mother, primarily through the placenta (Sternfeld, 1997). Since severe hyperthermia has been associated with teratogenic effects, particularly neural tube defects, and since the primary

determinant of foetal temperature appears to be maternal temperature, maternal overheating during exercise may induce adverse foetal outcomes via foetal hyperthermia (Sternfeld, 1997; Shephard, 2000; Borg-Stein *et al*., 2005; Dempsey *et al*., 2005; Morris & Johnson, 2005).

It is assumed that a safety mechanism exists which offers a degree of protection against hyperthermia. Studies have demonstrated that when exercising during pregnancy, the maximum maternal temperature reached is lower than pre-pregnancy values (Dempsey *et al*., 2005; Morris & Johnson, 2005). These changes are first seen early in the first trimester and are further increased as pregnancy continues. By the 7th week of pregnancy, the maximal maternal temperature during exercise falls by 0.3^{o}C and continues to further decrease by 0.1^{o}C for each month of gestation. At term, the maximum maternal temperature is substantially decreased (by 70%) compared to pre-pregnancy values. In addition, and probably contributing to the latter, the temperature at which sweating begins abruptly falls by the 7th week of gestation and continues to decrease throughout pregnancy (Morris & Johnson, 2005). Two possible mechanisms may contribute to this lower level of thermal stress. Firstly, the increased blood volume associated with pregnancy, and secondly the possibility of a reduction in the sweating threshold. Both of these will allow evaporative heat loss to occur at a lower body temperature. In addition, the foetus itself may have mechanisms to protect itself from thermal stress (Shephard, 2000; Morris & Johnson, 2005). These thermoregulatory adaptations appear in early pregnancy and therefore may confer a protective effect during the critical period of foetal development and limit the thermal stress in women who continue to exercise throughout pregnancy (Morris & Johnson, 2005). Although current studies suggest that efficient maternal thermoregulatory processes during pregnancy protect against hyperthermia (Clapp, 1996; Sternfeld, 1997; Riemann & Kanstrup-Hansen, 2000; Dempsey *et al*., 2005), it would be irresponsible to allow exercise to raise maternal temperature to any significant extent.

Birth weight

Several authors have reviewed the influence of regular recreational exercise on birth weight. It can be concluded that there is no consensus in the literature (Clapp, 1996; Hale & Milne, 1996; Sternfeld, 1997; Riemann & Kanstrup-Hansen, 2000; Magann *et al*., 2002; Dempsey *et al*., 2005; Morris & Johnson, 2005). Possible reasons for the discrepancies can be due to: small sample sizes, selection bias, pre-pregnancy fitness, trimester in which the exercise is performed, absence of adequate controls, differences in study populations, study designs, types of exercise, duration of exercise, maternal eating habits and the statistical analysis of the data (Hale & Milne, 1996; Sternfeld, 1997; Clapp, 2000; Magann *et al*., 2002; Morris & Johnson, 2005). It is worth noting that even in studies where there was decreased birth weight among exercising women, the babies were still within the normal birth weight range (Sternfeld, 1997).

EXERCISE AND LABOUR/DELIVERY

Preterm labour/delivery/birth

There has been some concern as to whether training can induce preterm labour (Clapp, 2000; Riemann & Kanstrup-Hansen, 2000). The concern is because of the effect of the prostaglandins produced at the uterine neck combined with the rise in catecholamines during exercise (Riemann & Kanstrup-Hansen, 2000). Results regarding exercise during pregnancy and the incidence of preterm labour are conflicting (Morris & Johnson, 2005). Several studies have shown no increase- in risk for preterm labour in women who exercised during pregnancy (Hatch, Levin, Shu & Susser, 1998; Alderman, Zhao, Holt, Watts, & Beresford, 1998; Riemann & Kanstrup-Hansen, 2000; Rao, Kanade, Margetts, Yajnik, Lubree, & Rege, 2003; Morris & Johnson, 2005; Hegaard, Damm, Nielsen & Pedersen, 2006), with some even indicating a decreased risk with strenuous exercise (Clapp, 1996; Dempsey *et al*., 2005). In a prospective study, supporting the latter possibility, it was found that women in the moderate energy expenditure group had fewer incidences of pre-labour rupture of membranes, while women in the lower energy expenditure category had increased risks

for preterm birth. However, most of the differences were explained by the characteristics of the women in each expenditure level rather than the exercise pattern itself (Magann, Evans & Newnham, 1996). It has been shown that women, who continue weight-bearing exercise at or above training level, benefit by having their babies five to seven days earlier than those who stop exercising before the 28th week (Clapp, 1996; Clapp, 2000). As a result significantly more women, who continue to exercise, deliver by their "due date". This timing is viewed, by women, as a distinct advantage (Clapp, 1996).

Another concern, previously expressed, is the possibility of exercise-induced uterine irritability that may result in premature uterine contractions and preterm delivery. Investigators once reasoned that maternal physical activity results in a metabolic milieu consistent with an increase in the synthesis and release of prostaglandins and noradrenaline (Sternfeld, 1997; Dempsey *et al*., 2005). Prostaglandins and noradrenaline are potent uterine stimulants capable of inducing premature contractions. Studies have failed to support these concerns (Clapp, 1996; Hale & Milne, 1996; Sternfeld, 1997; Dempsey *et al*., 2005). Although exercise does increase uterine contractility transiently (Clapp, 1996), the magnitude is small (Hale & Milne, 1996) and does not necessarily lead to increased incidence of preterm labour (Clapp, 1996). It is further indicated that neither commencing with, nor continuing regular exercise regimens during pregnancy, increase the incidence of membrane rupture before the onset of labour at term (Clapp, 1996; Hale & Milne, 1996; Magann, Evans & Newnham, 1996).

Labour and delivery

Various studies have shown that women who exercise during pregnancy derive several benefits during labour and delivery periods including: shorter labours (Sternfeld, 1997; Wang & Apgar, 1998; Clapp, 2000; Riemann & Kanstrup-Hansen, 2000; Magann *et al*., 2002; Morris & Johnson, 2005), less complicated labours (Clapp, 1996; Clapp, 2000; Riemann & Kanstrup-Hansen, 2000; Hegaard *et al*., 2006), and less complicated deliveries (Clapp, 2000; Riemann & Kanstrup-Hansen, 2000),

less strenuous deliveries (Henriksson-Larsen, 1999), increased normal spontaneous vaginal deliveries (Clapp, 1996; Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005), decreased need for obstetric/medical intervention (Clapp, 1996; Wang & Apgar, 1998; Clapp, 2000; Morris & Johnson, 2005), decreased forceps deliveries (Clapp, 2000; Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005), decreased Caesarean Section rates (Sternfeld, 1997; Clapp, 2000; Borg-Stein *et al*., 2005; Riemann & Kanstrup-Hansen, 2000; Morris & Johnson, 2005) and decreased cord abnormalities (nuchal cord and true knot in cord) (Clapp, 1996; Morris & Johnson, 2005). Unfortunately, it seems that most of these benefits are not seen for women who start or continue with low-intensity, non-weight-bearing regimens or for those who decrease their exercise performance substantially during pregnancy (Clapp, 2000). In contrast to the above, Mangann *et al*. (2002) found that women who exercise excessively, are more likely to need an induction of and have longer first-stage labours (time from the onset of labour to complete cervical dilatation) resulting in longer total labour time. Variations in the outcomes of the different studies are generally related to pre-pregnancy fitness levels, the intensity, type and duration of exercise, and the controls used in the studies (Morris & Johnson, 2005). It would appear that babies born of regularly exercising mothers tolerate labour well, have an increased tolerance for the physiologic stresses of late pregnancy and show less behavioural or biochemical evidence of undue stress during labour (Clapp, 2000).

EXERCISE AND THE POSTPARTUM PERIOD

Neonatal/Foetal outcomes

Data from studies that evaluated foetal outcomes at birth indicate that sustained exercise during pregnancy is associated with similar (Magann *et al*., 2002) or better outcomes than those observed with inactive mothers or mothers that discontinued exercise during pregnancy (Hale & Milne, 1996; Sternfeld, 1997; Henriksson-Larsen, 1999; Riemann & Kanstrup-Hansen, 2000; Borg-Stein *et al*., 2005; Dempsey *et al*., 2005; Morris & Johnson, 2005).

The offspring of exercising mothers would appear to manifest fewer signs of stress during delivery and are usually characterized by a better general condition as reflected in higher Apgar scores (Hale & Milne, 1996; Sternfeld, 1997; Henriksson-Larsen, 1999; Riemann & Kanstrup-Hansen, 2000; Magann *et al*., 2002; Borg-Stein *et al*., 2005; Morris & Johnson, 2005). Adverse neonatal outcomes are thus not considered to be increased for exercising women (Davies, Wolfe, Mottola, MacKinnon, Arsenault & Bartellas, 2003; Leiferman & Evenson, 2003).

Most types of recreational exercise are associated with a great deal of low frequency vibration (foot strike). The human foetus readily perceives vibrations in utero. Ongoing neurodevelopmental studies indicate that these recurrent stimuli are perceived by the foetus and seem to alter its behavioural responses to environmental stimuli after birth. It would appear that the newborn offspring of exercising women orientate themselves better to environmental sound and visual stimuli, are more alert, less cranky and self-quiet with greater ease after disruptive stimuli. Although unproved, it is said that these behavioural differences may be the result of the recurrent foetal behavioural stimulation associated with maternal exercise (Clapp, 1996; Clapp, 2000).

Recovery after parturition

Pregnant women who exercise regularly recover more quickly from the stresses and strains of parturition (Clapp, 2000; Lochmuller & Friese, 2005). It has been suggested that women exercising throughout pregnancy can half the time required for postpartum recovery (Clapp, 1996).

Lactation

It appears that moderate exercise during lactation does not in itself negatively affect the quantity or composition of breast milk, nor impact the growth of the infant (Hale & Milne, 1996; Davies, Wolfe, Mottola, MacKinnon, Arsenault & Bartellas, 2003; Borg-Stein *et al*., 2005). The observed reduced milk production may be caused by an inadequate fluid intake due to the fact that the exercising mother may not be aware of the need to increase her fluid intake to compensate

for the demands of the infant, as well as with those of exercise (Hale & Milne, 1996). The energy demands of regular exercise do not appear to impede lactation. Indeed, comparisons suggest that relative to their sedentary peers, women who choose to exercise regularly secrete larger volumes of milk with higher energy content. This benefit may reflect enhanced levels of serum prolactin or in relevant conditions, a relief in postpartum depression (Shephard, 2000).

A few years later

Women who continue to exercise throughout pregnancy and the postpartum period seem to have a more relaxed maternal-child relationship (Clapp, 2000). It has been shown that, compared to matched controls, at one year, the offspring of women who exercised during pregnancy exhibit slightly better motor skills, but have similar mental skills and morphometry. However, they seem to be much leaner than control offspring and to perform much better on standardized tests of intelligence, particularly in the area of language skills at age five years. It is anticipated that these children will remain lean and intellectually advantaged and will have improved cardiovascular and metabolic function, improved insulin sensitivity and diminished long-term cardiovascular risk when they are young adults (Clapp, 1996, Clapp, 2000).

RECOMMENDATIONS FROM LITERATURE

From the literature, the overriding consensus seems to be that there is very little increase in the risk of harmful effects with responsible exercising during pregnancy (Sternfeld, 1997; Wang & Apgar, 1998; Sundgot-Borgen, 2000; Ross & Brundage, 2002; Davies *et al*., 2003; Lochmuller & Friese, 2005; Borg-Stein *et al*., 2005; Hegaard *et al*., 2006). In fact, current evidence shows that in most cases, both the mother and the foetus benefit substantially when the mother starts or continues a regular exercise programme during pregnancy (Clapp, 1996; Clapp, 2000; Riemann & Kanstrup-Hansen, 2000; Shephard, 2000; Frey, 2002; Borg-Stein *et al*., 2005; Dempsey *et al*., 2005). Perhaps all healthy, well-nourished women, with uncomplicated pregnancies and no medical contra-indications, should be encouraged to participate in a regular

exercise regimen during their pregnancy (Sternfeld, 1997; Wang & Apgar, 1998; Sundgot-Borgen, 2000; Ross & Brundage, 2002, Davies *et al*., 2003; Lochmuller & Friese, 2005; Borg-Stein *et al*., 2005; Hegaard *et al*., 2006), as the benefits seem to outweigh the risks (Margolis, 1996; Clapp, 2000). However, caution should be taken to avoid exercise in extreme environmental conditions, especially under conditions of high temperatures and humidity (Sternfeld, 1997; Riemann & Kanstrup-Hansen, 2000). The million dollar question would therefore be: – "How much exercise and what type of exercise, at the various stages of pregnancy, is feasible without compromising her own or her foetus's health?"

Current recommendations

For women who have not been exercising regularly before pregnancy, it is deemed safe to commence an exercise programme (Sternfeld, 1997; Borg-Stein *et al*., 2005). The *Canadian Clinical Practice Guidelines* for exercise during pregnancy and the postpartum period recommends that previously sedentary women should be advised to start with 15 minutes of moderate intensity, continuous exercise three times per week and to work towards a goal of 30 minutes, four times per week (Borg-Stein *et al*., 2005). Women whose pregnancy is complicated with medical or obstetric problems should seek medical advice for specific individualized exercise recommendations (Borg-Stein *et al*., 2005).

Women previously active in regular recreational sport, prior to conception, may continue their exercise programme (Sternfeld, 1997; Borg-Stein *et al*., 2005) depending on the prepregnancy type and intensity of exercise. According to Shepard (2000), light to moderate activity of 30 minutes duration, four times a week, can safely be pursued during the early stages of pregnancy, but the quantity of exercise performed should not be increased over previous levels during the first 14 and the final 12 weeks of pregnancy. The *American College of Obstetricians and Gynaecologist* (ACOG) guidelines for exercise during pregnancy and the postpartum period recommends 30 minutes or more of moderate- intensity exercise per day for most days of the week during pregnancy

in the absence of medical or obstetric complications (Committee on Patient Education of the ACOG, 2003).

For competitive athletes engaged in strenuous sports, close medical supervision is recommended due to non-availability of conclusive data (Borg-Stein *et al*., 2005).

Research evidence on exercise during pregnancy could probably be summarized by saying that:

> a regular frequency of exercise is preferable to sporadic physical activity (Sternfeld, 1997),
>
> the level of physical activity should be appropriate to each woman's level of fitness and may need to be either increased or decreased at different stages of pregnancy (Clapp, 1996),
>
> that the level of fitness and activity before pregnancy is the main determinant of exercise during pregnancy (Borg-Stein *et al*., 2005).

Precautions/Concerns

According to current opinions in literature the following precautions should be taken into consideration when a woman continues or starts an exercise programme during pregnancy:

> A physician should screen for any contra-indications to exercise (Wang & Apgar, 1998).
>
> Caution should be taken in cases where there is a history of spontaneous abortion or premature labour (Shephard, 2000).
>
> Twin pregnancies require a more limited schedule of physical activity (Shephard, 2000).
>
> Adequate hydration is of utmost importance (Clapp, 1996; Sternfeld, 1997; Wang & Apgar, 1998; Riemann & Kanstrup-Hansen, 2000).
>
> Appropriate ventilation, in order to prevent possible teratogenic effects due to overheating, is of great importance (Wang & Apgar, 1998; Borg-Stein *et al*., 2005).
>
> Activities in extreme environmental conditions should be avoided, especially high temperatures and humidity (Sternfeld, 1997; Riemann & Kanstrup-Hansen, 2000).

Exercise that involve risks for the following should be avoided:

- abdominal trauma (Sternfeld, 1997; Wang & Apgar, 1998; Riemann & Kanstrup-Hansen, 2000; Borg-Stein *et al*., 2005).
- falling /loss of balance (especially in 3rd trimester) (Sternfeld, 1997; Wang & Apgar, 1998; Shephard, 2000; Davies *et al*., 2003). As the pregnancy reaches the 5th to 6th month, there is a progressive lordosis of the vertebral column associated with a forward tilt of the pelvis. This results in a change in the centre of gravity and a tendency for the exerciser to pitch forward. In stop-and-go exercise regimens, this tendency can be a potential source of injury (Hale & Milne, 1996).
- excessive joint stress/ sudden maximal joint movement (Sternfeld, 1997; Wang & Apgar, 1998; Riemann & Kanstrup-Hansen, 2000).

Other recommendations include:

avoid exercise in the supine position after the first trimester since the weight of the baby may interfere with proper blood circulation (Sternfeld, 1997; Wang & Apgar, 1998; Riemann & Kanstrup-Hansen, 2000).

avoid long periods of standing (Clapp, 1996; Wang & Apgar, 1998).

ensure adequate caloric and nutrient intake (Sternfeld, 1997; Wang & Apgar, 1998).

strength training and isometric muscle contractions to maximal intensity are generally not recommended because these can cause an increase in blood pressure (Riemann & Kanstrup-Hansen, 2000).

avoid weight lifting, horseback riding and strenuous anaerobic exercises (Greydanus & Patel, 2002).

Signs and Symptoms which indicate that exercise should be reassessed or discontinued

Exercise should be reassessed or discontinued with the presence of the following symptoms/signs: vaginal bleeding (Wang & Apgar, 1998; Riemann & Kanstrup-Hansen, 2000; Shephard, 2000; Borg-Stein *et al*., 2005), chest pain (Wang & Apgar, 1998; Borg-Stein *et al*., 2005), severe shortness of breath, sudden severe abdominal pain (Wang & Apgar, 1998), preterm labour, premature ruptured membranes, pregnancy-induced hypertension (Shephard, 2000; Borg-Stein *et al*., 2005), incompetent cervix (Riemann & Kanstrup-Hansen, 2000; Shephard, 2000; Borg-Stein *et al*., 2005), intrauterine growth retardation, failure of normal foetal weight gain, placental injury or dysfunction (Shephard, 2000), dyspnoea prior to exertion, dizziness, headaches, muscle weakness, decreased foetal movement, amniotic fluid leakage (Borg-Stein *et al*., 2005), signs suggestive of pre-eclampsia (swelling of extremities, headaches, dizziness or disturbances of vision) (Borg-Stein *et al*., 2005); (Riemann & Kanstrup-Hansen, 2000; Shephard, 2000), and uterine contraction that continues for more than 30minutes after exercising (Wang & Apgar, 1998; Shephard, 2000). Exercise-induced uterine contractions are usually more related to the type of exercise than the intensity (Riemann & Kanstrup-Hansen, 2000). A study on uterine activity during the third trimester, with equivalent intensities, showed cycling to lead to uterine activity in 50% of sessions, running in 40%, rowing in 10% and recumbent cycling in 0% (Durak, Jovanovic-Peterson & Peterson, 1990). In contrast, Veille (1985) noted no increase in uterine activity with moderate maternal exercise (running and stationary cycling) during the last 8 weeks of pregnancy. Absolute contra-indications to aerobic exercise include: hemodynamically significant heart disease, restrictive lung disease, multiple gestation at risk for premature labour (Borg-Stein *et al*., 2005) and persistent second-or third- trimester bleeding (Greydanus & Patel, 2002).

Types of exercise

The following types of exercise are recommended for women during pregnancy:

Swimming (Sternfeld, 1997; Wang & Apgar, 1998; Clapp, 2000; Lochmuller & Friese, 2005): Swimming (in appropriate environmental conditions) is recommended as the safest form of exercise during pregnancy (Clapp, 2000). It is especially recommended given the support of body mass and the easier control of core temperatures (Shephard, 2000).

Aquatic exercises e.g. aquarobics, running in water (Sternfeld, 1997; Shephard, 2000): Aquatic exercise is reported to decrease back pain intensity, control peripheral oedema and to result in a lower heart rate response during the exercise. Furthermore buoyancy has the effect of unloading joints to ease painful movements (Borg-Stein *et al*., 2005).

Stationary cycling (Wang & Apgar, 1998; Clapp, 2000; Shephard, 2000; Lochmuller & Friese, 2005).

Walking (Sternfeld, 1997; Wang & Apgar, 1998).

Low-impact aerobics (Sternfeld, 1997; Wang & Apgar, 1998; Lochmuller & Friese, 2005). Specialized aerobic exercise programmes for pregnant women exist.

Running (Wang & Apgar, 1998; Lochmuller & Friese, 2005). Care should be taken with regard to intensity, duration and pregnancy characteristics, as running increases the likelihood of musculoskeletal injury due to increased laxity of connective tissue (Sternfeld, 1997).

In addition to building overall muscular strength and endurance, the goals for muscle conditioning in prenatal programmes are to improve posture, provide support for breasts, strengthen muscles used during labour and prevent urinary incontinence. Specific activities that accomplish these goals include shoulder shrugs and rotations, pelvic tilts and rocks, abdominal curls and kegels (Sternfeld, 1997). The pelvic tilt and pelvic floor muscle training during pregnancy was shown to prevent urinary incontinence during pregnancy and after

delivery. It has also been found to be useful in alleviating round ligament pain and to decrease back pain intensity (Sternfeld, 1997; Borg-Stein *et al*., 2005). Exercise to increase strength of the abdominal and back muscles is also recommended (Borg-Stein *et al*., 2005).

Physical activities that involve exposure to changes in environmental pressure may negatively influence gestation. For instance the ACOG guidelines for exercise during pregnancy and postpartum period recommend that pregnant women should avoid scuba diving (Committee on patient education of the ACOG, 2003). This recommendation is supported by the fact that women who dive recreationally to levels requiring decompression on a regular basis, show evidence of a three to six fold increase in the incidence of spontaneous abortion and congenital malformation, as well as an increased incidence of foetal growth restriction and preterm labour (Clapp, 2000). It has also been shown that the rates of pregnancy complications are much higher and birth weights lower at altitudes above 3333m, which suggests that exposure to the additional physiologic stress produced by exercising at high altitudes may not be wise (Clapp, 2000).

Postpartum exercise

It would appear that recumbent abdominal and pelvic floor exercises, along with deep breathing, static muscular contractions and light walking can be initiated soon after delivery, but intensive abdominal exercises should be deferred until abdominal muscle separation is corrected. Individual tolerance provides the best guide to the progression of exercise (Shephard, 2000). More than 90% of women who maintain an exercise regimen during pregnancy continue to exercise after parturition and of these, 70% reach or exceed their pre-pregnancy fitness levels. The average postpartum time, after which women begin exercise, is at the end of the second week, but it generally ranges from three days to eight weeks (Hale & Milne, 1996; Clapp, 2000). Exercise intensity usually returns to pre-pregnancy levels by the sixth month and at that time, their maximal aerobic capacities exceed their pre-pregnancy level by 6-15%.

Nine out of 10 of these women appear neither to experience pain, heavy bleeding, nor musculoskeletal, reproductive or breastfeeding problems associated with the early resumption of postpartum exercise. These women also seem to regain their abdominal muscle tone rapidly (Clapp, 2000). Currently there are no known maternal contra-indications for the athlete who has been exercising throughout her pregnancy to resume exercise in the immediate postpartum period. It has even been shown that world class athletes, have trained and competed in world championships, within 3 months, postdelivery (Hale & Milne, 1996).

IN SUMMARY

The value of current studies is limited because there are large differences among studies in the type, intensity, duration and frequency of the exercise regimens and the time during pregnancy when the studies were carried out (Wiswell, 1996; Clapp, 2000). The difference in sample size (Magann *et al*., 2002), pre-pregnancy fitness levels, inconsistent use of control groups (Morris & Johnson, 2005), as well as several sources of potential bias, also make the different studies difficult to compare.

In general, literature does not support the concerns associated with exercise during normal uncomplicated pregnancies, but rather shows that both the mother and the foetus benefit when the mother exercise during pregnancy. For example, women who exercise during pregnancy show a greater sense of well-being, fewer symptoms of pregnancy (e.g. nausea, swelling etc), increased maximal aerobic capacity, increased maternal blood volume and increased cardiac output. However, it seems that these benefits are limited in women who do not continue to exercise throughout their whole pregnancy. Exercise has also been shown to stimulate placental growth and improve foetal oxygen and substrate delivery at maternal rest. Currently there is no consensus among authors on the influence of regular exercise on birth weight. A major concern has been the possibility of teratogenic effects associated with hyperthermia. However, all current studies report an enhanced maternal thermoregulation that partially protects against hyperthermia.

There are conflicting results regarding exercise during pregnancy and the incidence of preterm labour. Although it has been found that exercise increases uterine contractility transiently, the magnitude is small and generally does not lead to an increased incidence of preterm labour. The offspring of women who exercised regularly during pregnancy manifest fewer signs of stress during delivery and have higher Apgar scores than the offspring of women who do not exercise during pregnancy.

In general, it appears that women who exercise during pregnancy have shorter and less complicated labours, uncomplicated deliveries, increased normal spontaneous vaginal deliveries and that they need fewer medical interventions. The energy demands of regular exercise do not appear to impede lactation. Indeed, comparisons suggest that relative to their sedentary peers, women who choose to exercise regularly secrete larger volumes of milk with higher energy content.

CONCLUSION

At present, indications are that all women with uncomplicated pregnancies should be encouraged to participate in a regular suitable exercise regimen. Swimming, in appropriate environmental conditions, is recommended as the safest form of exercise during pregnancy if the women ensure adequate hydration, good ventilation and sufficient caloric and nutrient intake. Exercises that involve the risk of falling, abdominal trauma and excessive joint stress, should be avoided.

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