

**NUTRITION AND MATERNAL-FETAL EXCHANGE OF NUTRIENTS:
A DIFFERENT PERSPECTIVE***

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SUMMARY

The assumption that the fetus can parasitize a malnourished mother is still widely accepted. In contrast with this concept, data reviewed here demonstrate that during a reduced availability of energy and protein the mother protects her body stores of nutrients at the expense of sustaining a normal rate of fetal growth. The mechanisms by which the mother prevents fetal parasitism would involve a reduced blood volume expansion, reduced placental blood flow and, ultimately, reduced transfer of nutrients. Whenever maternal nutritional status is not optimal some degree of fetal growth retardation tends to occur. In a large population this is reflected in a downward shift in mean birth weight and increased incidence of low birth weight infants with associated increase in neonatal mortality.

INTRODUCTION

Nearly four decades ago it was proposed that circulating nutrients are distributed between the maternal tissues and the conceptus, according to their respective metabolic rates.¹ Since the feto-placental unit was supposed to have a higher metabolic rate than any of the maternal tissues, except the brain, it was assumed that during a reduced availability of nutrients the fetus would successfully compete for nutrients with the mother, thus becoming an effective parasite.

The assumption that the fetus can parasitize a malnourished mother is still

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widely accepted and it has fostered the idea that only extreme degrees of maternal malnutrition can produce significant degrees of growth retardation, especially in humans. However, the results of recent studies and the reinterpretation of data published in the last two decades have raised serious doubts about the validity of past assumptions on fetal parasitism and, in general, on the nature of maternal-fetal interactions during malnutrition. The purpose of this review is to analyze this new evidence and to discuss both its biological and clinical implications.

STUDIES IN ANIMAL MODELS

The hypothesis that the division of nutrients between the mother and the fetus is based solely on metabolic rates was tested in pregnant rats kept on a food-restricted diet or injected with growth hormone to increase their food intake.² The results showed that whether the food intake of the mother was increased, normal, or reduced, the increase in conceptus weight over the last half of pregnancy could be related to the total maternal weight increase over this period by the formula $y = 1/4 x + 4.5n$, where y is the combined weight of fetuses and placentas (conceptus weight), x is the total maternal weight increase over the last 11 days of pregnancy and n is the number of fetuses. The results of this study apparently confirmed the initial hypothesis of the division of nutrients since, as demonstrated by the formula, the increase or decrease of the total nutrients available to the mother had been shared equally between the various maternal tissues and the fetuses. An interesting finding of this study, however, deserves further analysis. The authors reported that the derived formula did not hold when the total maternal weight increase was less than 20 g, although they found that even in the most restricted animals the conceptus weight never fell below $4.5(n+1)$.

Studies in rats food restricted between the first and the 20th day of gestation suggest, however, that a more severe food restriction may cause the conceptus weight to fall markedly below the $4.5(n+1)$ limits.³ Furthermore, in these circumstances, the conceptus weight as calculated by the formula discussed above, is higher than the observed weights, suggesting that in severely food-restricted rats the division of nutrients does not favor the conceptus over the mother. For example, rats fed only 25 percent of the quantity of food fed the control group had a mean fetal weight of 1.89 g. Assuming a proportional reduction in placental weight and 1.5 ml of amniotic fluid per fetus the total conceptus weight would have been approximately 48 g. The weight of the mothers, after removal of the conceptus, was 148 g. Adding the weight of the conceptus, it can be assumed that the final total maternal weight was 196 g. This would represent a loss of 4 g of maternal initial body weight. Applying the formula discussed above the weight of conceptus should have been approximately 56 g, which is 17 percent higher than the real weight as calculated above.

The apparent anomaly in the distribution of the available nutrients between the mother and the fetus is also observed when proportional reduction in maternal body weight, conceptus weight, and litter size in rats suffering increasing degrees of food restriction are compared.³ Thus, in rats restricted to 75 percent of their *ad*

libitum intake, the mothers gained 9 g of net body weight (after removing the conceptus and the uterus) while the mean fetal weight was maintained within control values. When mothers were restricted to 50 percent of their normal intake, they lost 8 percent of their initial weight while the mean fetal weight decreased by approximately 12 percent. In addition, 23 percent of the animals had embryonic deaths and subsequent reabsorption of the fetuses. When mothers were restricted to 25 percent of their normal intake, they lost 26-32 percent of their initial body weight, 64-92 percent had embryonic deaths, and in two studies only 8 percent of the mothers were able to produce litters of 12-14 pups. In these rats mean fetal weight was reduced to 46-56 percent of control weights. The data suggest that the food-restricted pregnant rat is able to sustain fetal growth within normal limits only when the food restriction allows her to gain some weight. If the food restriction is more severe, however, both litter size and fetal growth are reduced disproportionately to the loss of maternal body weight.

As shown in Table 1 the apparent disproportion between the magnitude of maternal losses in body weight and the deficit in the body weight of the fetuses at term described in the rat, also occurs in other mammalian species when the mother is fed either a calorically-deprived or a protein-deprived diet. For example, in the pregnant guinea pig, food restriction or a low-protein diet causes a 50 percent fetal mortality and a 22-28 percent reduction in birth weight of the surviving fetuses while

TABLE 1
MATERNAL BODY WEIGHT AND BIRTH WEIGHT CHANGES CAUSED BY
FOOD RESTRICTION OR PROTEIN RESTRICTION IN VARIOUS
MAMMALIAN SPECIES

Species	Type of restriction	Percent change in body weight		
		Mother*	Fetus	Ref.
Rat	50% _o food intake	-10	-12	(3)
	75% _o food intake	-32	-51	(3)
Guinea pig	60% _o food intake	-12	-22	(4)
	low-protein diet	-10	-28	(4)
Pig	low-protein diet	-16	-33	(5)
Macaca Mulatta	low-protein diet	- 1	-10**	(6)
	low-protein diet	- 7***	-15	(8)

* Compared with preconceptional weight.

** Reduction found not significant.

*** Data taken from a graph.

the mothers lose 10-12 percent of their initial body weights.⁴ Pigs fed a low-protein diet during pregnancy lost 17 percent of their initial body weight while the body weight of the fetuses at term was 33 percent below control values.⁵

Studies in Rhesus monkeys in which pregnant mothers fed a diet providing 1 g of protein per kg of body weight per day were compared with mothers fed 4 g of protein/kg/day have shown that the protein-deprived mothers maintain a normal energy intake and normal fetal growth while losing approximately 1 percent of their initial body weight.⁶ The results were attributed to maternal metabolic adaptations to a low-protein diet.⁷ Other studies in Rhesus monkeys also fed diets providing 1 g of protein/kg/day have shown an increase in maternal and perinatal mortality and a 15 percent reduction in mean birth weight when liveborn and stillborn fetuses were computed together.⁸ In spite of some of the discrepancies in the results of the studies conducted in the Rhesus monkeys, they suggest that given an adequate caloric intake the mother is able to metabolically adapt to a low protein intake and sustain fetal growth apparently more successfully than in energy-deprived animals. In other species, such as the rat, a low protein diet during gestation causes a reduced food intake and, therefore, a superimposed energy deficit. Thus, a reduced availability of energy may be more deleterious to fetal growth than a reduced availability of protein.

As clearly shown by the experiments conducted in rats, the division of nutrients between the mother and the fetus seems to be influenced by different mechanisms according to the availability of nutrients. As expressed by the formula $y = 1/4 x + 4.5n$ if enough nutrients are available since early pregnancy fetal needs are fully met and if the maternal food intake increases above normal a portion of the excess nutrients also goes to the fetus. Thus, the fetus is apparently given unlimited access to the available nutrients. However, when food intake is restricted, and the availability of nutrients reach a critical level, the fetus is not favored over the mother. On the contrary, the deviation of nutrients seems to favor maintenance of maternal body weight over maintenance of a normal rate of fetal growth. If the restriction is severe enough to induce maternal body weight losses, obviously the fetus becomes an active parasite but, in contrast with past assumptions, it does not receive a proportionally larger share of nutrients than the mother. This new concept of maternal-fetal relationships concerning the division of nutrients is strongly supported by the results of refeeding experiments of previously restricted pregnant rats.³ As shown in Table 2, rats restricted to 25 percent of their normal intake from the first up to the fifth day of pregnancy and then fed *ad libitum* have a final body weight and mean fetal weight similar to controls. If refeeding is started only in the last 12 days of pregnancy the mother increases her net body weight (after removal of the conceptus and uterus) by 23 percent (versus 28 percent of controls) while the fetuses show a significant 7 percent deficit in body weight. If refeeding is started on the last 10 days the mothers gain 21 percent of net body weight and the fetuses show a significant 12 percent deficit in body weight. These experiments clearly show that when nutrients become available after a period of restriction the mother is greatly favored over the fetus. Obviously, if active fetal parasitism was taking place during the period of food restriction, it seems logical to assume that the fetus would have been favored over the mother when nutrients again become available.

TABLE 2
EFFECT OF UNRESTRICTED FEEDING ON MATERNAL AND FETAL BODY WEIGHT AFTER VARYING INITIAL PERIODS OF 75 PERCENT DIETARY RESTRICTION³

Days of restriction (from conception)	% Change in maternal body weight*	Mean fetal weight (g)
5	+27	4.01
7	+23	3.75
9	+21	3.53
11	+19	3.39

* Compared with body weight at conception.

Recent studies on the effect of food restriction on the body composition of pregnant and non-pregnant rats have provided new insights into the nature of maternal fetal interactions during a reduced availability of nutrients. This study⁹ showed that at term pregnant rats fed 50 percent of the food consumed by control animals had a similar body composition of pair-fed non-pregnant rats while the mean body weight of the fetus was significantly reduced (Table 3). These results clearly demonstrate

TABLE 3
CARCASS COMPOSITION IN CONTROL AND 50 PERCENT FOOD RESTRICTED PREGNANT AND NON-PREGNANT RATS^{1,2}

Group	n	Water %	Fat %	Lean dry tissue %
Pregnant control	5	60.03 ^a ± 0.74	17.3 ^a ± 0.5	22.7 ^{a,b} ± 0.6
Non-pregnant control	5	63.07 ^b ± 0.6	13.0 ^b ± 0.7	23.9 ^a ± 0.3
Non-pregnant restricted	4	65.81 ^b ± 0.68	9.07 ^c ± 1.2	24.9 ^a ± 0.7
Pregnant restricted	4	65.74 ^b ± 0.51	9.00 ^c ± 0.5	25.2 ^{a,c} ± 0.4

1 Weight of the samples was representative of their groups.

2 Mean ± S.E.M. Values not sharing a common superscript letter (s) in the same column are significantly different ($P < 0.05$ or less).

that the pregnant food-restricted rat is not parasitized by the fetus. In addition, they suggest that important metabolic adjustments must occur to allow the mother to prevent this parasitism. Comparisons of food efficiency, as determined by changes in body weight per quantity of food ingested, between the pregnant and non-pregnant animals revealed that during pregnancy there is a marked increase in food efficiency. For control rats the increased food efficiency was apparent throughout pregnancy while in the malnourished rats it was apparent only during the last 10 days of gestation.

STUDIES IN HUMANS

During the winter of 1944-45, several Dutch cities were exposed to a severe food shortage that lasted approximately 28 weeks. It has been estimated that the *per capita* caloric intake during that period of time dropped to a low of approximately 1,200 kcal/day.¹⁰ Many women were pregnant at the time of the famine and, although it is conceivable that they may have received some extra food from family members, their food intake must have fallen considerably short of their needs. An analysis of the effects of famine on birth weight¹⁰ revealed a significant decrease of approximately 250 g, compared with pre-famine values for the entire population, among the women affected during the second and third trimester of gestation. This seemingly small difference of approximately a ten percent reduction in birth weight caused by conditions considered extreme has been interpreted as an indication that the human fetus is also an effective parasite or, conversely, that the mother is able to "adapt" to a reduced food intake to sustain fetal growth. Further, considering that famine conditions during the last two trimesters caused only a 250 g fall in mean birth weight, the extrapolation has been made that marginally-deficient diets should produce a negligible effect on birth weight.¹¹

A question that is rarely considered, however, is the effect that pregnancy under famine conditions had in the mothers. No data has been made available on the pre-pregnancy weight and changes in body weight of these women. It is, therefore, more difficult to compare in severely food-restricted humans the type of changes in maternal body weight and birth weight, discussed in the previous section for food-restricted rats. However, since the postpartum weight of the Dutch women measured in the 9-10th day after delivery has been reported, it is possible to make some gross estimates of their body weight losses using some reasonable assumptions. The first assumption, considering that the average weight of a young adult female in an industrialized country is 56 kg, is that the average body weight of a Dutch woman during the pre-famine period and following many months of food rationing was 55 kg or less. The mean postpartum weight during the pre-famine period was 59 kg. Subtracting from this weight an estimated 3 kg due to extra body fluids, enlarged breasts and uterus, the women that delivered during the pre-famine period had a minimum of 1 kg of extra body stores. If the same factors are subtracted from the postpartum weight of women that suffered famine during various periods of pregnancy (Table 4) it is found that the women most affected by famine may have lost approximately an average of 1.5 kg of their initial body weight. This is less than 3 percent of their initial

TABLE 4

**ESTIMATED CHANGES IN POSTPARTUM MATERNAL BODY STORES
AND CHANGES IN MEAN BIRTH WEIGHT IN DUTCH WOMEN
BEFORE AND AFTER FAMINE CONDITIONS**

	Postpartum maternal weight (kg)	Δ Body stores	Birth weight (g)
Pre-famine	59.0	+ 1.0	3,338
Famine during 3rd trimester	57.6	- 0.4	3,220
Famine during 2nd and 3rd trimester	56.5	- 1.5	3,011
Famine during 1st and 2nd trimester	61.0	+ 3.0	3,370
Famine during 1st trimester	61.5	+ 3.5	3,312
Post-famine	62.0	+ 4.0	3,308

body weight. Since in this group the reduction in mean birth weight is approximately 10 percent compared with pre-famine values, it is obvious that the mothers most affected by the famine were proportionally less affected than their infants. In this regard, food-restricted women would behave similarly to other food-restricted mammals, suggesting the possibility that in spite of obvious biological differences, certain basic principles concerning maternal-fetal exchange during food restriction may be common to several mammalian species.

The disproportionate effect that small changes in maternal body weight may have on birth weight are also evident in the studies conducted in a large number of individuals in which maternal pre-pregnancy weight and weight gain during pregnancy have been correlated with birth weight. These studies have shown that both pre-pregnancy weight and weight gain during pregnancy can influence birth weight independently of each other. For example, high pre-pregnancy weights or high weight gains during pregnancy are associated with higher mean birth weights. In contrast, low pre-pregnancy weights or low weight gains during pregnancy are associated with lower mean birth weights. When the variables change in the same direction, for example, a high pre-pregnancy weight is associated with a high weight gain during pregnancy, they tend to have an additive effect on birth weight. These variables can

also neutralize each other's effects; thus, a low pre-pregnancy weight followed by a high pregnancy weight may result in a mean birth weight similar to a high pre-pregnancy weight followed by a low weight gain during pregnancy.^{1,2} The effect on mean birth weight of some of these combinations are shown in Table 5. It must be stressed, however, that maternal body weight or weight gain during pregnancy are only one of the many variables that can influence the rate of fetal growth. Thus, in contrast with the animal models, correlation between maternal weight changes and birth weight become apparent in humans only when large numbers of cases are analyzed.

TABLE 5

**JOINT RELATIONSHIP OF PRE-PREGNANCY WEIGHT
AND MATERNAL WEIGHT GAIN WITH BIRTH WEIGHT
IN 622 TERM PREGNANCIES IN WHITE WOMEN,
JOHNS HOPKINS HOSPITAL, BALTIMORE, 1954-1961^{1,2}**

Maternal group	Mean birth weight (g)	% of low birth weight infants
High pre-preg. weight, high weight gain	3,831	0.0
High pre-preg. weight, low weight gain	3,628	2.3
Low pre-preg. weight, high weight gain	3,453	1.5
Low pre-preg. weight, low weight gain	3,044	5.8

Definitions: high pre-pregnancy is > 72 kg (160 lb); high weight gain is > 13 kg (20 lb); low pre-pregnancy weight is < 54 kg (120 lb); low weight gain is < 4 kg (9 lb).

The changes in both pre-pregnancy weight and weight gain during pregnancy are reflected in the maternal postpartum weight. Preliminary results of a study including 254 singleton term deliveries from uncomplicated pregnancies^{1,3} has shown that mean birth weight increases linearly with increasing postpartum weights until postpartum weight is 100-110 percent of maternal ideal body weight. At that point birth weight remains constant while postpartum weight continues to increase (Fig. 1). This correlation suggests that optimal fetal growth occurs only when the mother is able to accumulate a critical amount of extra body stores during pregnancy. While

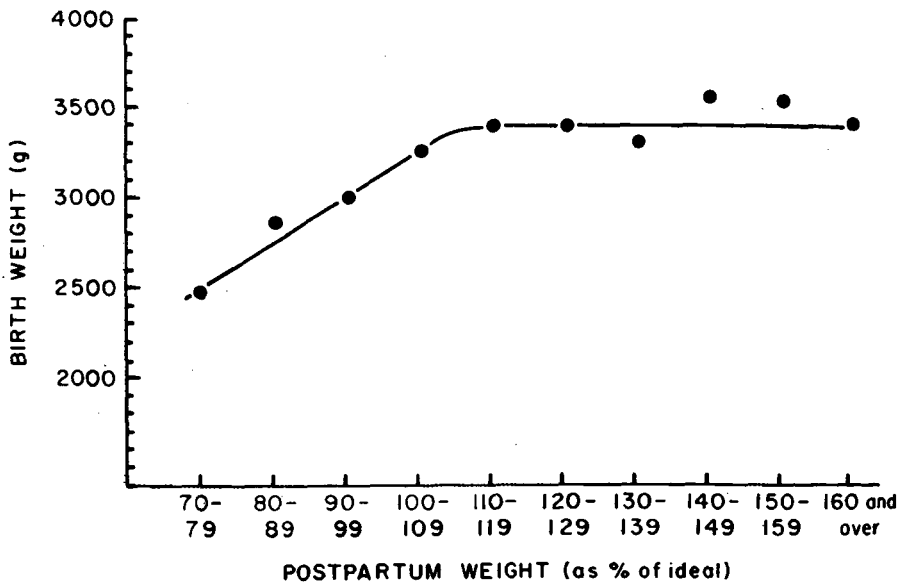


Fig. 1. Gestational changes in maternal stores, plasma volume, placental weight, fetal weight and uterine blood flow in human pregnancy. Values are expressed as percent of maximal increments. Data derived from Hytten and Leitch.¹¹

accumulation of body stores beyond what seems to be desirable does not have any further positive effects on birth weight, it is evident that when the mother fails to accumulate extra body stores the rate of fetal growth begins to decrease progressively. Again, as observed in the animal models, the evidence clearly contradicts the concept that the fetus is protected by the mother when nutritional status is less than optimal or that the fetus can protect itself by parasitizing the mother. Further, the uneven distribution of available nutrients that occurs when food-restricted rats are allowed unrestricted amounts of food may also occur in humans. This is suggested by a correlation between birth weight and maternal weight gain in women with low pre-gravid weight.¹⁴ This correlation demonstrates that for each 100 g increments in mean birth weight the mother gains 2.3 kg of body weight. Again, this observation seems to conflict with the idea of active fetal parasitism.

MALNUTRITION AND MATERNAL-FETAL EXCHANGE

The animal and human data clearly demonstrate that in contrast with prevailing concepts, the undernourished mother does not adapt to sustain fetal growth but rather to maintain her own body stores of nutrients. An important role seems to be played by the quantity of maternal body stores of energy. If the mother has extra quantities of these stores she can maintain a normal rate of growth even if her food intake is inadequate. If the body stores are small, then food intake has a crucial role in maintaining fetal growth.

Teleologically, the fact that among eutherian mammals nature protects the mother more than the fetus seems reasonable from the point of view of survival of the species. For example, during a famine caused by a serious drought a normal size newborn delivered by a nutritionally-depleted mother would have little chance to survive if the mother cannot initiate lactation, cannot defend herself and the young, or cannot cover enough distance during the day to search for food. A stronger female that produces a runt or a smaller litter probably has a better chance to survive and conceive again the following season.

The metabolic and physiological mechanisms used as adaptive response by the food-restricted mother are still largely unknown. In protein-restricted rats the rate of urea synthesis and the levels of certain hepatic enzymes involved in amino acid catabolism are reduced.^{15,16} Reports of a reduced urea excretion in women receiving low-protein diets¹⁷ suggests that similar mechanisms to reduce urinary nitrogen losses are also present in humans. The results, previously discussed, on Rhesus monkeys fed low-protein diets can also be considered as indirect evidence of major adaptations in protein and amino acid metabolism.⁶⁻⁸ The nature of these changes in amino acid metabolism, however, still remain elusive. Similarly, the mechanisms responsible for the increased food efficiency observed in pregnant rats⁹ are also unknown. Probably many factors, including the changes in amino acid metabolism and the degree of physical activity, are involved. All these possibilities, however, require further investigation.

During normal pregnancy a great number of physiological adjustments take place. Obviously all these adjustments are either directly or indirectly involved in sustaining fetal growth. Among the earliest and best known changes are the expansion of blood volume and the deposition of maternal stores of nutrients, presumably mostly fat. As shown in Figure 2, in humans these changes are occurring at a maximal rate at midgestation and therefore they precede by several weeks the period of maximal placental and fetal growth. They also precede the period of maximal increase in the rate of uterine blood flow which coincides with the peak of fetal growth. It is tempting to speculate that this sequence represents an orderly chain of events in which only the adequate completion of the earlier maternal changes allows the adequate completion of the final ones involving the growth of the conceptus. For example, a woman who receives an adequate diet can both deposit adequate amounts of extra stores and also expand blood volume. Obviously, blood volume expansion is a process influenced by many variables besides nutrition. The need to expand red cell mass and to a lesser extent circulating protein mass, must depend,

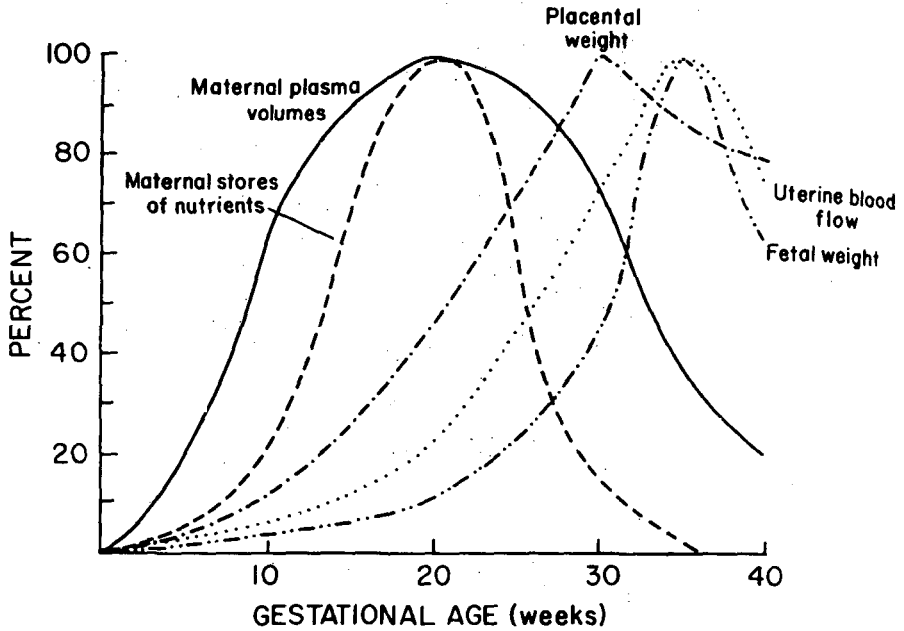


Fig. 2. Mean birth weight in 254 singleton term deliveries in women with different postpartum weights.¹⁴

however, on the adequate availability of substrate and energy. The need for an expand blood volume during gestation is not entirely clear, but it is conceivable that it may be necessary to compensate for the major hemodynamic changes caused by the expansion of the uterine blood flow. In the rat as well as in humans and other species uterine blood flow increases many-fold during the course of pregnancy.^{18,19} In order to maintain this high rate of blood flow to the uterus without reducing the rate of blood flow to other organs an expansion of blood volume and an increased cardiac output would appear to be an appropriate compensatory mechanism. Consequently, if blood volume expansion early in pregnancy is inadequate, uterine blood flow may also be inadequate to sustain optimal fetal growth. A correlation between these two factors has not been established yet. It has been reported, however, that maternal plasma volume expansion correlates with the birth weight of the infants.²⁰

In the rat either a 50 percent food restriction or a protein restriction from day 5 of gestation produced a significant reduction in the degree of expansion of plasma volume.²¹ Most affected were the protein-restricted animals. When plasma volume was expressed per 100 g of net maternal body weight no differences were found

between controls and food-restricted rats indicating that both plasma volume expansion and the deposition of maternal stores were equally dependent on the level of food intake. Still, the protein-restricted rats had significantly lower values than any of the other groups suggesting that the availability of certain amino acids may have an important role in plasma volume expansion (Table 6).

In more recent experiments blood flow to the kidney, the uterus and the placenta has been measured in food-restricted rats.²² Results demonstrate that compared with control animals, food-restricted rats have a marked reduction in the rate of blood flow to the uterus and the placenta while maintaining adequate blood flow to the kidneys. These data support the idea that plasma volume/blood volume expansion is necessary to adequately expand uterine and placental blood flow and that no redistribution of blood flow from other vital organs occurs as an alternative adaptation.

The reduced placental blood flow most likely reduces the availability of nutrients to the fetus. This possibility is supported by earlier studies demonstrating that protein-restricted rats have a reduced rate of transfer of glucose and amino acids from the maternal circulation into the fetus.^{23, 24} A reduced maternal-fetal transfer of nutrients in the malnourished animals not only helps to explain the fetal growth retardation of the restricted mothers but it may also explain why the fetus is not able to actively parasitize the maternal stores.

CONCLUSIONS

The consequences of maternal malnutrition during pregnancy have been the focus of considerable controversy in the past. As pointed out in a recent review of this subject, an important source of controversy has been the results of studies in which obvious flaws of design and data analysis can be recognized.²⁵ One of the arguments most frequently used against the importance of adequate nutritional management during pregnancy, involving active counselling and, when necessary, food supplements has been that the mother can "adapt" to protect the fetus and that the fetus is an active maternal parasite. Therefore, at least in humans, no harm to the fetus will result from moderate inadequacies in the maternal nutritional status. The evidence presented in this review clearly demonstrates that these ideas are unfounded, as is the theory of the division of nutrients from which they originated.

The importance of maternal nutrition on the outcome of pregnancy becomes obvious when the focus of attention is shifted from the possible consequences in a single individual to a large population. As shown by perinatal mortality curves, a "small" downward shift in mean birth weight increases disproportionately the mortality rates. In the U.S., for example, the black population has a mean birth weight that is 150 g lower than the white population and a perinatal mortality twice as high.²⁵ One of the factors influencing the higher mortality rates is the higher proportion of low birth weight infants that results from a shift to the left in mean birth

TABLE 6
MATERNAL PLASMA VOLUME (PV) IN PREGNANT AND NON-PREGNANT RATS FED
VARIOUS DIETS DURING PREGNANCY*

Group	n	PV (ml)	PV per 100 g of total body weight	PV per 100 g of net body weight	PV per 100 g of conceptus
Standard diet	15	16.96 ± 0.61 ^a	4.48 ± 0.18 ^a	5.91 ± 0.10 ^a	24.68 ± 1.01 ^a
50 % restricted	17	13.14 ± 0.34 ^b	4.70 ± 0.16 ^a	6.03 ± 0.14 ^a	22.32 ± 0.71 ^a
25% casein	15	17.91 ± 0.58 ^a	4.84 ± 0.21 ^a	5.95 ± 0.17 ^a	27.83 ± 0.89 ^b
6% casein	11	10.50 ± 0.34 ^c	3.70 ± 0.10 ^c	4.70 ± 0.17 ^b	19.57 ± 0.93 ^c
Non-pregnant (standard diet)	10	10.94 ± 0.35 ^c	—	4.14 ± 0.11 ^c	—

* Means are given ± S.E.M. Values not sharing a common superscript letter in the same column are significantly different ($P < 0.05$ or less).

weight. The opposite happens when the mean birth weight of a population shifts upwards. This has been clearly demonstrated in a rural Guatemalan population when the increase in mean birth weight associated with a higher caloric intake provided by a food supplement decreased by 50 percent the number of low birth weight babies and associated mortality.^{2,6}

Although the incidence of maternal malnutrition is closely associated with the prevailing socio-economic conditions of a country, there is ample evidence that even the most affluent nations are not immune to the problem. As shown here, and in contrast with past assumptions, seemingly small inadequacies in maternal nutritional status can significantly affect fetal growth. The realization of this fact should make adequate care of the nutritional needs of the pregnant mother an integral part of comprehensive prenatal care.

RESUMEN

NUTRICION E INTERCAMBIO MATERNO-FETAL DE NUTRIENTES: UNA PERSPECTIVA DIFERENTE

La suposición de que el feto puede parasitar a la madre desnutrida continúa siendo un concepto de amplia aceptación. En contraste, datos revisados en el presente trabajo demuestran que ante una disponibilidad reducida de energía y proteínas, la madre protege sus reservas orgánicas de nutrientes a expensas del sostenimiento de una tasa normal de crecimiento del feto. Los mecanismos por los que la madre evita el parasitismo fetal implicarían una menor expansión del volumen sanguíneo, disminución del flujo sanguíneo placentar, y la transferencia reducida de nutrientes. Siempre que el estado nutricional de la madre deja de ser óptimo, tiende a ocurrir un retardo del crecimiento fetal. En una población grande esto se refleja en una tendencia a declinar del promedio del peso al nacer, y en una mayor incidencia de niños con bajo peso al nacer asociada a un aumento de mortalidad neonatal.

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