Chronic maternal dietary iodine deficiency but not thiocyanate feeding affects maternal reproduction and postnatal performance of the rat

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Iodine deficiency disorders affect reproductive performance in the afflicted populations. Environmental iodine deficiency (ID) and goitrogens are important in their aetiology. We observed earlier that chronic maternal dietary ID but not goitrogen feeding altered the blood-brain barrier nutrient transport in adult rats. Whether similar differences exist in their effects on reproduction of dams and postnatal performance of the offspring has been assessed. Inbred, female, weaning WNIN rats were rendered hypothyroid by feeding for 8-12 weeks, a low iodine test diet or a control diet with added potassium thiocyanate (KSCN) (@ 25 mg/rat/day). Following mating with control males, they continued on their respective diets till their pups were weaned. Indices of reproductive performance such as percentage of conception, mortality of dams during pregnancy and parturition, litter size, and survival of pups till weaning were affected markedly by ID but not thiocyanate feeding. Neither ID nor thiocyanate feeding from conception or parturition affected their reproductive performance. Nevertheless, postnatal weight gain of pups was less in all the three ID groups but not thiocyanate fed dams. Rehabilitation of chronically ID pregnant dams from conception or parturition did not improve their pregnancy weight gain, litter size or birth weight of pups but decreased abortion and mortality of mothers during pregnancy and parturition. Rehabilitation improved the pups’ postnatal weight gain but the effect was only moderate. Based on the results of the present study it may be suggested that maternal ID but not thiocyanate feeding affects reproductive performance and postnatal performance of their offspring.

Keywords: Goitrogens, Hypothyroidism, Iodine deficiency, Reproduction, Thiocyanate

Dietary iodine deficiency (ID) and environmental goitrogens important in the aetiology of IDDs, are believed to exert their influence through changes in the thyroid status (hypothyroidism)¹. Thyroid insufficiency and reproductive failure have been reported earlier in iodine deficient farm animals². In areas of iodine deficiency, development of the foetus was retarded or arrested at some stage in gestation resulting in early death or resorption-abortion or stillbirth³. Also, the offspring were weak, hairless and associated with prolonged gestation, parturition and retention of placental membrane⁴. Potter et al.⁵ have demonstrated in iodine deficient sheep, a significant loss of lambs due to abortion and stillbirth as compared to controls. Sub-clinical hypothyroidism in humans often causes infertility⁶,⁷, which may be restored after thyroxin therapy⁸,⁹.

However, earlier in WNIN adult rats, marked differences between goitrogens (mild vs potent)¹⁰,¹¹ and iodine deficiency¹²,¹³ in their effects on body weight and blood-brain barrier nutrient transport were observed. The differences were observed despite the fact that both chronic ID and potassium thiocyanate feeding produced hypothyroidism of comparable intensity. Taken together with the foregone literature, these observations prompted us to hypothesize that maternal dietary iodine deficiency and goitrogens may have different effects on their reproductive performance and/or the survival/postnatal performance of the offspring.

The present studies have been carried out essentially to validate/negate this hypothesis. In addition, the stage of development at which ID/thiocyanate feeding would have greater impact on these parameters as well as the reversibility of changes by rehabilitation from different points such as conception and parturition have been determined.

Materials and Methods

Iodine deficient diet (Low iodine test-LIT) was procured from M/S ICN Pharmaceuticals Inc, UK. Iodine content of the control and LIT diets were 3 mg
and 22.8 μg respectively per kg of diet. Radio-immuno assay (RIA) kit for T₄ was purchased from BRIT, BARC, Mumbai, India and all other chemicals used were of analytical grade procured locally. Deionized, glass distilled water was used for feeding the experimental animals.

The protocol used for all the animal experiments described below was approved by the Institute’s Animal Ethics Committee (IAEC), which ensures that the regulations laid down by the Governmental Agency in this regard are complied with strictly.

Effect of chronic iodine deficiency/potassium thiocyanate feeding on mortality, morbidity and reproductive performance of Wistar/NIN (WNIN) rat dams—The protocol used in these studies for feeding the experimental animals of different groups is depicted schematically in Fig. 1. Inbred, female, weaning Wistar/NIN (WNIN) rats were assigned to one of the three groups and fed as follows: group 1-LIT diet sufficient in all nutrients except iodine (E1); group 2-iodine sufficient control diet (C) and group 3-iodine sufficient control diet with added potassium thiocyanate (@ 25 mg/rat/day) (E1)¹⁰. They were housed individually in polypropylene cages with wire mesh bottom and maintained at 22° ± 2°C, under standard lighting conditions (12: 12 light/dark cycle). They were fed their respective diets ad libitum till the experimental diet (LIT diet or control diet with added KSCN) fed animals (E1) became hypothyroid (serum T₄ < 2-3 μg/dl) (i.e., 12 weeks in case of ID and 8 weeks with thiocyanate feeding). Body weights of the animals were monitored regularly and their thyroid status assessed by determining the levels of serum thyroxine (T₄) and urinary iodine excretion¹⁴.

After confirming their hypothyroidism, rats were subjected to vaginal smear tests to check the regularity of their estrus cycles. At the pro-estrus stage, they were mated with normal adult male rats and the day on which the sperm appeared in vaginal smear was considered as the day one of gestation. Except when stated otherwise, pregnant rats received their respective diets throughout pregnancy and lactation (till the postnatal day 21 of their offspring). Body weight of the pregnant dams was determined on day 14-15 of gestation and throughout the gestation period, dams in all the four groups were examined for morbidity and mortality. Number of mothers delivering successfully and their litter size were noted in all the four groups. In addition, survival of the pups and their body weight gain till weaning was also monitored in different groups. Also, the wet weight of their brains was recorded at the time of their killing i.e, on postnatal day 21.

Effect of ID/potassium thiocyanate feeding of varying initiation points and duration—To determine the stage of development at which the dams were more vulnerable to the effects of ID or thiocyanate feeding, a group of control pregnant dams were shifted to LIT or thiocyanate diet from conception (E2) and another group from parturition (E3). The morbidity and mortality of the live born pups until weaning were monitored as mentioned above.

Effect of rehabilitation/potassium thiocyanate withdrawal—To evaluate the prevention/reversibility of the changes induced by dietary ID/thiocyanate feeding on the parameters mentioned above, groups of pregnant ID/thiocyanate fed dams were shifted to their respective control diet either from conception (R1) or parturition (R2) and the parameters mentioned above were monitored.

Statistical analysis—Data were analysed statistically by the appropriate use of one-way analysis of variance (ANOVA) coupled with critical difference test¹⁵.
Results

Effects of maternal ID/potassium thiocyanate feeding on morbidity, mortality and reproductive performance of WNIN rat dams—At the end of twelve weeks of feeding LIT diet, the levels of serum T4 [2.46 ± 0.86 (E1) vs. 5.36 ± 1.13(C) (μg/dl)] (P < 0.05) and urinary iodine excretion [1.276 ± 0.592 (E1) vs. 2.312 ± 1.373 (C) (μg/mg creatinine)] were lower in ID dams as compared to controls indicating that the rats were iodine-deficient/hypothyroid. Also, their weight gain during pregnancy was significantly lower than that of controls (P < 0.001) (Table 1).

Conception rate was 75% of controls in the chronically ID (E1) group and unlike controls, there were abortions (20%), death of dams during pregnancy (15%) and parturition (15%) in the ID dams. While 100% of control pregnant dams delivered live offspring, this was only 50% in the ID dams. Further, the litter size was lower in ID dams than the controls. However, no such effects were observed in the control dams fed LIT diet from conception (E2) or parturition (E3) (Table 1).

Chronic thiocyanate feeding (for eight weeks from weaning) produced a moderate degree of hypothyroidism in the dams comparable to that in ID dams (serum T4 2.31 ± 0.53 μg/dl). However, unlike in the chronic ID (E1) dams, the urinary excretion of iodine was significantly (P < 0.05) higher (8.90 ± 3.60 μg/mg creatinine) in thiocyanate fed rats than controls (3.31±1.49 μg/mg creatinine) suggesting higher levels of circulating iodide in these rats than controls. Nevertheless, there was no effect of any of the three dietary thiocyanate feeding regimes, on any of the parameters of maternal reproduction mentioned above (Table 2).

Performance of the pups born to control, ID and thiocyanate fed dams—The litter size was smaller and cannibalism higher in ID dams than controls, but the birth weight of pups born to chronically ID (E1) dams was comparable to those born to control dams (Table 3). Nevertheless, to obliterate the effects of different litter sizes during lactation, five pups per mother were maintained during lactation in both control and ID dams. In spite of this, weaning body weight of E1 pups was markedly lower that that of controls and the survival of the pups till weaning was only around 30% in chronically ID (E1) pups as compared to 100% in controls.

Although the birth weight of pups born to control (C) and iodine deficient: E2 and E3 pups was comparable, the weaning body weight of E2 and E3 pups was lower than that of controls and comparable to that of E1 pups. The wet weight of the brain (at weaning) per 100 g body weight was significantly

<table>
<thead>
<tr>
<th>Parameters</th>
<th>C</th>
<th>E2</th>
<th>E3</th>
<th>E1</th>
<th>R1</th>
<th>R2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight at mating (g)</td>
<td>158 ± 14.2</td>
<td>158 ± 14.2</td>
<td>158 ± 14.2</td>
<td>100 ± 14.8*</td>
<td>100 ± 14.8</td>
<td>100 ± 14.8</td>
</tr>
<tr>
<td>(40)</td>
<td>(40)</td>
<td>(40)</td>
<td>(45)</td>
<td>(45)</td>
<td>(45)</td>
<td></td>
</tr>
<tr>
<td>Body weight at 2 weeks of gestation</td>
<td>176 ± 4.80</td>
<td>163 ± 3.27</td>
<td>180 ± 3.90</td>
<td>123 ± 3.69*</td>
<td>124 ± 4.64</td>
<td>127 ± 6.29</td>
</tr>
<tr>
<td>(9)</td>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td></td>
</tr>
<tr>
<td>Mated animals which conceived (%)</td>
<td>100</td>
<td>-</td>
<td>-</td>
<td>75</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Conceived mothers which aborted (%)</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>20</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Conceived mothers dead during pregnancy (%)</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>15</td>
<td>15.4</td>
<td>None</td>
</tr>
<tr>
<td>Conceived mothers dead during delivery (%)</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>15</td>
<td>7.68</td>
<td>7.60</td>
</tr>
<tr>
<td>Conceived mothers which delivered (%)</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>50</td>
<td>53.8*</td>
<td>61.5'</td>
</tr>
<tr>
<td>Litter size (No. of pups/mother)</td>
<td>8-10</td>
<td>8-10</td>
<td>8-10</td>
<td>3-5</td>
<td>3-5</td>
<td>3-5</td>
</tr>
</tbody>
</table>

C-Control diet through growth pregnancy and lactation, E1-LIT diet through growth pregnancy and lactation, E2-LIT diet from conception, E3-LIT diet from parturition, R1-E1 dams rehabilitated from conception and R2-E1 dams rehabilitated from parturition.

* The remainder of the pregnant dams did not deliver.

*P < 0.001 by One way ANOVA/critical difference (E1 vs C)
higher (than controls) in the pups of the three ID groups: E1, E2 and E3.

On the other hand, none of the three thiocyanate feeding regimes had any such effects on the pup’s birth-weight, survival and performance till weaning or on the wet weight of their brains per unit body weight at weaning (Table 4).

Effect of rehabilitation/thiocyanate withdrawal—Thiocyanate feeding of any duration from any point of initiation had no effect on maternal mortality, morbidity, reproductive performance or survival/postnatal performance of the offspring. As such, its withdrawal from the dam’s diet, either from conception (R1) or parturition (R2) had no effect (Tables 2 and 4) on any of these parameters. Rehabilitation of chronically iodine deficient pregnant dams from conception (R1) did not improve the weight gain or death of dams during pregnancy, % of pregnant dams which delivered or the litter size but prevented the abortions and partly decreased the death of dams during parturition (Table 1). R1 had no effect on the birth weight of pups, but the percentage of pups that survived till weaning was improved significantly by both the rehabilitation regimens R1 and R2 (Table 3). Although the weaning body weight of R1 and R2 pups was higher than that of ID (E1, E2 or E3) pups, they weighed significantly lower than controls at weaning (Table 3). The wet weight of the brain per unit body weight (at weaning) was lower in R1 and R2 pups than in ID (E1, E2 or E3) pups, but they were significantly higher ($P < 0.001$) than that of control pups.

### Table 2—Effect of KSCN feeding/withdrawal on mothers reproductive performance

<table>
<thead>
<tr>
<th>Parameters</th>
<th>C</th>
<th>E2</th>
<th>E3</th>
<th>E1</th>
<th>R1</th>
<th>R2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight at mating (g)</td>
<td>154 ± 8.28</td>
<td>154 ± 8.28</td>
<td>154 ± 8.28</td>
<td>168 ± 12.3</td>
<td>168 ± 12.3</td>
<td>168 ± 12.3</td>
</tr>
<tr>
<td>Body weight during second week of pregnancy</td>
<td>190 ± 15.8</td>
<td>190 ± 15.8</td>
<td>190 ± 15.8</td>
<td>210 ± 13.8</td>
<td>210 ± 13.8</td>
<td>210 ± 13.8</td>
</tr>
<tr>
<td>Mated animals conceived (%)</td>
<td>100</td>
<td>-</td>
<td>-</td>
<td>100</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Conceived dams which aborted (%)</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Conceived dams dead during pregnancy (%)</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Litter size</td>
<td>7-9</td>
<td>6-9</td>
<td>7-9</td>
<td>6-8</td>
<td>7-8</td>
<td>7-8</td>
</tr>
</tbody>
</table>

C-Control diet through growth pregnancy and lactation, E1-Control diet with added KSCN (@25 mg/rat/day) through growth pregnancy and lactation, E2-Control diet with added KSCN from conception, E3-Control diet with added KSCN from parturition, R1-Dietary KSCN withdrawn from E1 rats from conception and R2-Dietary KSCN withdrawn from E1 dams from parturition, ND-None detected

### Table 3—Effect of dietary iodine deficiency/rehabilitation on the performance of the offspring

<table>
<thead>
<tr>
<th>Parameters</th>
<th>C</th>
<th>E2</th>
<th>E3</th>
<th>E1</th>
<th>R1</th>
<th>R2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight (g)</td>
<td>5.57 ± 0.097 (55)</td>
<td>4.78 ± 0.097 (56)</td>
<td>5.33 ± 0.073 (9)</td>
<td>4.9 ± 0.092 (28)</td>
<td>4.81 ± 0.09 (28)</td>
<td>5.3 ± 0.14 (29)</td>
</tr>
<tr>
<td>Live born pups dead postnatally (%)</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>69.5</td>
<td>23.1</td>
<td>16.7</td>
</tr>
<tr>
<td>Litter size maintained during lactation</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Live born pups which survived till weaning (%)</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>30.6</td>
<td>76.9</td>
<td>83.1</td>
</tr>
<tr>
<td>Weaning body weight (g)</td>
<td>29.4 ± 1.34a (28)</td>
<td>14.0 ± 0.66 (47)</td>
<td>13.6 ± 0.28 (16)</td>
<td>12.4 ± 0.48 (27)</td>
<td>18.1 ± 0.28 (26)</td>
<td>18.12 ± 0.53 (38)</td>
</tr>
<tr>
<td>Brain weight (g/100 g body weight) at weaning</td>
<td>4.42 ± 0.17b (22)</td>
<td>8.03 ± 0.34 (27)</td>
<td>7.51 ± 0.26 (6)</td>
<td>8.27 ± 0.37 (10)</td>
<td>6.44 ± 0.14 (20)</td>
<td>6.49 ± 0.20 (23)</td>
</tr>
</tbody>
</table>

a, b, P < 0.001, C vs all other groups by one way ANOVA/critical difference
Discussion

The estrus cycle of a thyroidectomized rat is prolonged and erratic after withdrawal of thyroid hormones\(^{16,17}\). In the present study, where hypothyroidism was induced by dietary iodine deficiency/thiocyanate feeding, no such disturbances were noticed in the estrus cycle. This could be due to the fact that the hypothyroidism produced in the present study was only moderate as evident from serum T4 levels (though lower in chronically ID/thiocyanate fed rats than controls) were higher than those found in thyroidectomized rats reported earlier\(^{16,17}\).

The lower rates of conception, higher mortality of dams during pregnancy and greater incidence of abortions and stillbirths in chronically iodine deficient (E1) dams are in accordance with previous literature that maternal iodine deficiency in the rat resulted in fewer viable embryos, whereas maternal thyroidectomy had a similar but more severe effect\(^4\). In the present study, morbidity was observed in some of the chronically iodine deficient (E1) dams and these observations are in general agreement with the findings in iodine deficient farm animals\(^5\).

Given that both ID and goitrogens are equally important in the IDD aetiology\(^{18,19}\), the finding that dietary thiocyanate feeding of any point of initiation or duration had no effect on any of the parameters studied [despite producing hypothyroidism of comparable magnitude as the ID (E1) group] was probably due to their different mechanism of action in different organs. To the best of our knowledge this is one of the first demonstrations of such differences between ID and goitrogens, \textit{vis a vis}, their effects on maternal reproduction and the pup’s performance. Although not comparable, these differences between ID and thiocyanate feeding in their effects on maternal reproductive performance are in line with earlier report that only chronic dietary ID\(^{13}\) but not thiocyanate feeding\(^{10}\) affected nutrient transport across the blood brain barrier in adult rats. The present results are at variance with those of Escobar \textit{et al}\(^4\) and Hetzel \textit{et al}\(^20\), who demonstrated that the effect of iodine deficiency disorders (specially those related to fetal development) are mediated through maternal hypothyroidism.

It is of interest that in the present study, only chronic maternal ID but not thiocyanate feeding affected the reproductive performance of rat dams although both produced hypothyroidism of comparable magnitude. It is known that the developing foetus derives its requirements of thyroid hormones from the mother during the first few weeks of gestation in the humans\(^{20,21}\) and till day 17-18 of gestation in the rat\(^4\). Later it has to synthesize its own thyroid hormones, for which it needs iodine and this obviously has to come from the mother. In chronic maternal dietary ID, this will be limiting because of the very low iodine content of the diet. On the other hand, in maternal thiocyanate feeding with no concurrent ID as in the present study, circulating iodide levels could be high in the mother due to the inhibition of thyroidal iodide uptake, stimulating iodide efflux or replacing iodide by thiocyanate resulting in an increase in the excretory level of iodine or iodine retaining capacity of the thyroid/body. This is dependent on the consumption pattern of thiocyanate arising from cyanogenic plant foods and its availability to the growing foetus may be better\(^22\).

The present finding that compared to controls, urinary...
excretion of iodine was significantly higher in mothers fed thiocyanate, while it was significantly lower in ID mothers appears to be in line with this inference.

The present observation that neither chronic maternal ID nor thiocyanate feeding affected the birth weight of the pups however seems to suggest that the moderate maternal hypothyroidism produced by these dietary regimes (with or without associated iodine deficiency in mothers) may be insufficient to affect foetal growth and hence the birth weight of the offspring. Chronic maternal ID itself at present level had no effect on the birth weight of the offspring and it appears reasonable that maternal ID of shorter duration eg, from conception (E2) had no effect on the birth weight of the offspring. That it (E2) had no effect even on maternal reproductive performance (which was affected by E1) could be due to insufficient duration of feeding for the animal to become iodine deficient. Since the mother was iodine sufficient at conception and probably for most part of gestation [at which several important events including organogenesis occur\(^2\)] feeding LIT diet from conception (E2) had no effect on the birth weight of the offspring. That it (E2) had no effect even on maternal reproductive performance perhaps failed to affect maternal reproductive performance. These results highlight the importance of maternal iodine deficiency early during gestation and suggest that the mother may have to be iodine deficient (not just moderately hypothyroid) right at conception, for any significant effect to be seen in the dam’s reproductive performance. This seems to be corroborated by our finding that rehabilitation of chronically ID (E1) dams from conception (R1) improved its reproductive performance \(albeit\) marginally.

Given the comparable degree of hypothyroidism in the offspring (at weaning) of ID/thiocyanate fed dams, it appears that the decreased weaning body weights only in the pups of ID but not thiocyanate fed mothers (regardless of the point of initiation of ID/thiocyanate feeding) could be due to higher level of iodide in circulation in thiocyanate fed (but not ID) dams and probably its greater availability to the offspring during lactation. That the postnatal performance of the offspring was affected only in all three ID groups but not the thiocyanate fed groups, again seems to stress the importance of maternal iodine deficiency rather than moderate hypothyroidism (with no concurrent iodine deficiency) in this effect. This appears to be further corroborated by our finding that a significant increase in brain wt/100 g body wt was observed in the pups of iodine deficient (E1, E2 and E3) and rehabilitated (R1 and R2) dams but not thiocyanate fed or rehabilitated dams. Indeed, the increase in brain wt/100 g body wt is seems to be due to a significant reduction in their body weights (40-60% of controls) due to dietary iodine deficiency rather than any change in the absolute weight of the brain. Further, the present finding that rehabilitation of ID (E1) dams from conception (R1) and parturition (R2) increased the postnatal survival of the pups but could not mitigate the effects on the pups’ body and brain weights (at weaning) to any significant extent, appears not only to highlight the importance of maternal iodine status during early intrauterine growth for the normal postnatal growth of the offspring and its brain but also may suggest that damage caused to the foetus and its brain due to maternal iodine deficiency early during pregnancy may not be reversible to a large extent. These findings are in partial agreement with evidence of similar nature in humans and experimental animals\(^2\).

It has been reported earlier that: (i) feeding potassium thiocyanate (@ 25 mg/rat/day) to WNIN rats through two generations also induced only moderate hypothyroidism (serum T\(_4\): 3.85 ± 0.73 vs 7.02 ± 1.50 μg/dl in controls) and significantly higher urinary iodine excretion (9.15 ± 3.45 μg/mg creatinine vs 3.05±0.65 μg/mg creatinine in controls) suggesting normal levels of circulating iodide in F1 dams (female offspring born to rat dams fed potassium thiocyanate throughout their growth, pregnancy, lactation; weaned on to and received the same diet throughout) and had no effect on their reproduction or postnatal performance of the F2 offspring (offspring born to F1 rat dams)\(^11\); (ii) Feeding potent goitrogens: propyl thiouracil\(^2\) or methyl mercaptoimidazole\(^11\) to pregnant WNIN rat dams induced severe hypothyroidism (serum T\(_4\): 0.65-0.67 μg/dl vs 4-6 μg/dl in controls) along with significantly lower excretion of urinary iodine (0.75-0.99 μg/mg creatinine vs 3-4 μg/mg creatinine in controls) suggesting low levels of iodide in maternal circulation and significantly decreased the birth weight of the offspring and their postnatal growth. Taken together all these results and the present findings indicate the importance of severe maternal hypothyroidism or moderate maternal hypothyroidism with concurrent dietary iodine deficiency (not without it) in modulating the reproductive performance of the
dams and the postnatal performance of their offspring. That administration of T3 to propyl thiouracil fed dams during lactation could restore their weaning body weight to that of control offspring appears to confirm the importance of severe hypothyroidism in regulating growth and reproduction\textsuperscript{25}.

Overall, the present studies have demonstrated for the first time to the best of our knowledge, significant differences between the two chief aetiological factors for IDD: thiocyanate (one of the common dietary goitrogens) and ID, in their effects on maternal reproduction and postnatal performance of the offspring. They indicate that moderate maternal hypothyroidism along with maternal iodine deficiency (but not without it) at conception/early gestation, but not later, adversely affect the reproductive performance of WNIN rat dams whereas iodine deficiency at any point of initiation affects the postnatal performance of the offspring. The adverse effects of chronic maternal ID on these parameters appear to be reversible albeit marginally.

Acknowledgement

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