

Chapter 13

Perinatal Taurine Depletion Increases Susceptibility to Adult Sugar-Induced Hypertension in Rats

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Abstract This study tests the hypothesis that perinatal taurine depletion produces autonomic nervous system dysregulation and increases arterial pressure in young male rats maintained on a high sugar diet. Sprague-Dawley dams were either taurine depleted (beta-alanine 3% in water) or left untreated from conception to weaning. Their male offspring were fed normal rat chow with or without 5% glucose. At 7–8 weeks of age, the male offspring were either tested in a conscious, unrestrained state or after anesthetia. Body weight was slightly lower in the taurine-depleted rats although their heart or kidneys to body weight ratios were similar. Plasma potassium, blood urea nitrogen, plasma creatinine, hematocrit, fasting blood glucose concentrations and glucose tolerance were all similar. In the taurine-depleted, high glucose group, mean arterial pressure and sympathetic nervous system activity were increased while baroreflex function was impaired. These findings suggest that in this model perinatal taurine depletion causes autonomic nervous system dysfunction that may contribute to dietary high sugar-induced hypertension.

Abbreviations *SHR*, spontaneously hypertensive rat; *CG*, control with glucose; *CW*, control without glucose; *TDW*, taurine depletion without glucose; *TDG* taurine depletion with glucose

13.1 Introduction

The perinatal environment can greatly influence adult function and disease development (Barker et al. 2002; Langley-Evans 2006). For instance, perinatal diets that are very low in nutritional value (e.g., proteins) can result in low birth weights in infants and subsequently insulin resistance, diabetes mellitus or arterial hypertension in adults (Barker et al. 2007; Eriksson et al. 2007). Mechanisms potentially underlying

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these changes include perinatal imbalances in circulating glucocorticoids, the renin-angiotensin system, oxidative stress and nephrogenesis. Substances that can inhibit or stimulate these factors either directly or indirectly (including perinatal angiotensin converting enzyme inhibitor treatment or taurine supplementation) can reduce these deleterious symptoms (Aerts and Van Assche 2002; Racasan et al. 2004; Wyss et al. 1994).

Taurine is a sulphur-containing beta-amino acid that plays many essential roles in prenatal and adult life (Sturman 1993), including intracellular volume regulation, cell membrane stabilization, neuromodulation, antioxidative stress, learning and memory, renal growth and differentiation, vasodilation and modulation of cardiac performance. Taurine is an essential amino acid during fetal life, a period in which there is limited endogenous biosynthesis (Aerts and Van Assche 2002). Maternal taurine supply to the fetus and the newborn can be severely limited due to abnormal maternal protein intake or an imbalance between protein and carbohydrate consumption (Barker et al. 2007; Forrester 2004; Langley-Evans 2006; Mendez et al. 2004). Though long-term effects of excess perinatal taurine exposure is not well established, perinatal taurine supplementation appears to be beneficial to the newborn (Chesney et al. 1998).

Previously, we have demonstrated that both prenatal and postnatal taurine depletion decrease renal blood flow and increase renal vascular resistance in adult, male rats (Roysommuti et al. 2004). In addition, both prenatal and postnatal taurine depletion significantly increase arterial pressure in adult, male offspring; however, they do not significantly alter natriuretic and diuretic responses to an acute intravenous saline load. Further, perinatal taurine depletion does not affect resting arterial pressure and heart rate. Many lines of evidence suggest that perinatal taurine depletion may increase an animal's sensitivity to risk factors of adult hypertension (Dawson et al. 1996; Schaffer et al. 2003).

High dietary carbohydrate intake is implicated in the pathogenesis of hypertension, and diets high in sugar increase arterial pressure in many animal models, including spontaneously hypertensive rats (SHR) and normotensive rats (Melancon et al. 2006; Reaven 1990; Shimamoto and Ura 2006). The underlying mechanism may involve the renin-angiotensin system, insulin resistance, sympathetic nerve activation, and renal damage (Roysommuti et al. 2002). The present study tests the hypothesis that perinatal taurine depletion leads to impairment of autonomic nervous system control of arterial pressure in normotensive, young adult, male rats. This impairment is exacerbated by high dietary sugar.

13.2 Materials and Methods

SD rats were bred in the animal unit of the Faculty of Medicine, Khon Kaen University and maintained at constant humidity ($60\pm 5\%$), temperature ($24\pm 1^\circ\text{C}$), and light cycle (0600–1800 h). Female SD dams were either taurine depleted (beta-alanine 3% in tap water, TD) or untreated (control, C) from conception to weaning. The

male offspring were fed normal rat chow with (TDG, CG) or without (TDW, CW) 5% glucose in their tap water throughout the experiment. All experimental procedures were preapproved by the Universities Animal Care and Use Committee and were conducted in accordance with the National Institutes of Health guidelines.

At 7–8 weeks of age, all male rats were placed under thiopental anesthesia and then implanted with femoral arterial and venous catheters. Two days later and after an overnight fast, arterial blood samples were obtained in the conscious animal for determination of Na, K, blood urea nitrogen (BUN), creatinine, hematocrit, and fasting blood sugar. Thereafter, glucose tolerance testing was initiated by an intravenous injection of glucose (2 g/kg in saline), and blood glucose levels were measured at 0, 30, 60, and 120 minute intervals. Twenty-four hours later, non-fasting blood samples were collected and then arterial pressure pulses were continuously recorded (Biopac system, CA) in the conscious animal before and during infusion of phenylephrine (increased arterial pressure) or sodium nitroprusside (decreased arterial pressure).

Renal nerve function was tested one day later in animals subjected to thiopental anesthesia. Renal sympathetic nerve function was assessed using stainless steel electrodes (12 MW, 0.01 Taper, A-M System, FL) connected to DAM80 amplifier (WPI, Sarasota, FL) and Biopac (Goleta, CA) system. Single unit recordings of renal nerve activity were conducted only on nerve units that responded to changes in arterial pressure following nitroprusside or phenylephrine infusion. Baroreflex sensitivity was measured as changes in heart rate and/or renal nerve activity per changes in mean arterial pressure.

Mean arterial pressure, heart rate, baroreflex sensitivity (BS) following phenylephrine or sodium nitroprusside and power spectrum densities of arterial pressure pulse were determined offline by using Acknowledge software 3.8.1 (Biopac, Goleta, CA). Plasma Na, plasma K, BUN, and plasma creatinine concentrations were measured by an automatic analyzer, hematocrit by a standard technique and blood sugar by standard glucostrips and a glucometer (Accu-chek[®], Germany). The autonomic nervous system control of arterial pressure was estimated from low frequency (0.3–0.5 Hz; sympathetic nerve activity) and high frequency (0.5–4.0 Hz; parasympathetic nerve activity) components of the power spectrum densities of baseline arterial pressure pulse using the Fourier analysis (Cerutti et al. 1991; Stauss and Kregel 1996).

All data were expressed as means \pm SEM. Statistical comparisons among groups ($p < 0.05$) were done by using one-way ANOVA and Duncan's Multi-Range (StatMost 3.6, Dataxiom Software, Los Angeles, CA).

13.3 Results

By 7–8 weeks of age, taurine depletion lowered body weight by about 10%; however, kidney and heart weights (as absolute weights or as ratios to body weight) were not significantly affected (Table 13.1). Further, fasting and non-fasting plasma potassium levels were not significantly different between the groups, while

Table 13.1 Body (BW), heart (HW), and kidney (KW) weights of male offspring

Treatment	BW (g)	KW (g)	HW (g)	KW/BW (%)	HW/BW (%)
CW (<i>n</i> = 6)	233 ± 4	1.12 ± 0.02	0.90 ± 0.02	0.48 ± 0.01	0.39 ± 0.01
CG (<i>n</i> = 5)	234 ± 7	1.13 ± 0.03	0.91 ± 0.02	0.48 ± 0.02	0.39 ± 0.02
TDW (<i>n</i> = 5)	210 ± 5*	1.07 ± 0.04	0.85 ± 0.02	0.51 ± 0.03	0.41 ± 0.01
TDG (<i>n</i> = 5)	214 ± 6	1.02 ± 0.06	0.89 ± 0.03	0.48 ± 0.04	0.42 ± 0.02

Data represent means±SEM. **P* < 0.05 when compared to CW. See text for abbreviations.

Table 13.2 Fasting and non-fasting plasma sodium and potassium of male offspring

Treatment	Plasma sodium (mEq/L)		Plasma potassium (mEq/L)	
	Fasting	Non-fasting	Fasting	Non-fasting
CW (<i>n</i> = 6)	139.8 ± 0.31	139.6 ± 1.15	3.72 ± 0.19	3.75 ± 0.19
CG (<i>n</i> = 5)	137.0 ± 2.09	138.6 ± 1.50	3.80 ± 0.22	3.82 ± 0.04
TDW (<i>n</i> = 5)	135.4 ± 1.81	139.4 ± 0.50	3.88 ± 0.07	3.86 ± 0.02
TDG (<i>n</i> = 5)	131.4 ± 2.38*	134.6 ± 2.11	3.68 ± 0.10	3.74 ± 0.18

Data represent means±SEM. **P* < 0.05 when compared to CW. See text for abbreviations.

non-fasting plasma sodium levels were not significantly different between the groups. In contrast, fasting plasma sodium concentrations were slightly, but significantly lower in TDG compared to all other groups (Table 13.2). Blood urea nitrogen (BUN) and plasma creatinine were unaffected by perinatal taurine depletion or dietary sugar supplementation (Table 13.3). Hematocrit, fasting blood glucose (Table 13.4), and glucose tolerance (Fig. 13.1) were also not significantly different between the groups. In contrast, all glucose treated animals exhibited elevated non-fasting blood sugar levels.

Neither dietary glucose supplementation nor perinatal taurine depletion of control animals altered mean arterial pressure or heart rate. However, perinatal taurine depletion followed by high dietary glucose significantly increased mean arterial pressure (Fig. 13.2). Power spectrum analyses of arterial pressure indicated that taurine depletion blunted both sympathetic and parasympathetic components. In control rats glucose supplementation did not affect either component, but in the taurine-depleted rats, glucose excess increased the sympathetic nerve component (Fig. 13.3). Further, in the taurine-depleted rats compared to all other groups, glucose decreased

Table 13.3 Fasting and non-fasting blood urea nitrogen and plasma creatinine

Treatment	Blood urea nitrogen (mg/dl)		Plasma creatinine (mg/dl)	
	Fasting	Non-fasting	Fasting	Non-fasting
CW (<i>n</i> = 6)	17.38 ± 0.75	18.17 ± 0.59	0.45 ± 0.02	0.43 ± 0.02
CG (<i>n</i> = 5)	16.86 ± 0.83	20.08 ± 0.96	0.46 ± 0.02	0.46 ± 0.02
TDW (<i>n</i> = 5)	16.72 ± 0.62	18.84 ± 1.02	0.46 ± 0.02	0.42 ± 0.02
TDG (<i>n</i> = 5)	18.02 ± 0.69	17.96 ± 0.91	0.44 ± 0.02	0.46 ± 0.02

Data represent means±SEM. No significant differences were observed between the groups. See text for abbreviations.

Table 13.4 Fasting (FBG) and non-fasting (NFBG) blood glucose and hematocrit of male offspring

Treatment	Blood glucose (mg/dl)		Hematocrit (%)	
	NFBG	FBG	Fasting	Non-fasting
CW (<i>n</i> = 6)	83.5 ± 2.9	80.3 ± 3.7	42.7 ± 0.7	42.2 ± 0.8
CG (<i>n</i> = 5)	105.6 ± 5.3*	80.8 ± 1.6	43.2 ± 0.6	42.2 ± 1.0
TDW (<i>n</i> = 5)	89.8 ± 5.6	81.2 ± 4.1	42.8 ± 0.7	41.4 ± 0.6
TDG (<i>n</i> = 5)	107.0 ± 4.9*	84.6 ± 3.4	43.4 ± 0.6	42.2 ± 0.8

Data represent means±SEM. * *P* < 0.05 when compared to CW. See text for abbreviations.

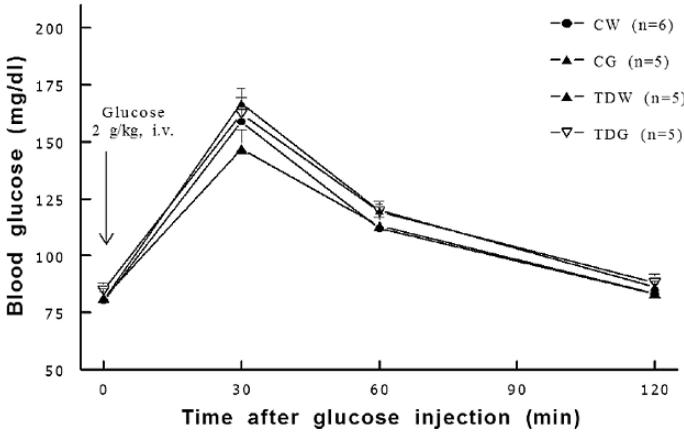


Fig. 13.1 All experimental groups displayed similar glucose tolerance (**P* < 0.05 to CW). See text for abbreviations

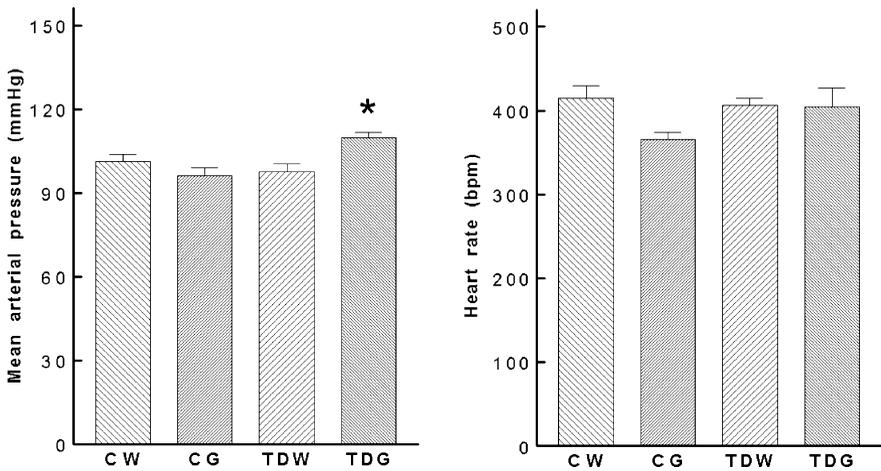


Fig. 13.2 High sugar intake increases mean arterial pressure in TDG while heart rate was not significantly different between the groups (**P* < 0.05 to CW). See text for abbreviations

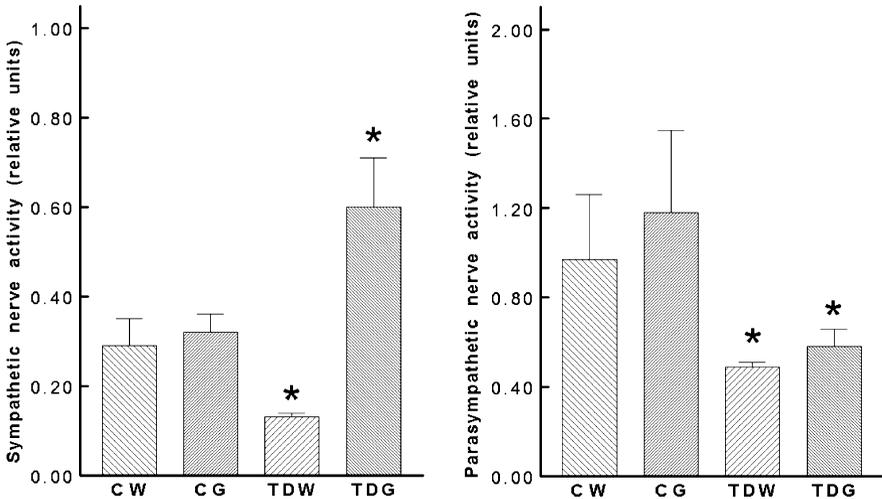


Fig. 13.3 Perinatal taurine depletion decreases both sympathetic and parasympathetic nerve activities in adult, male rats. High sugar intake heightened the sympathetic nerve activity only in the TDG (* $P < 0.05$ to CW). See text for abbreviations

the parasympathetic nerve spectral component of arterial pressure. Thus, the ratio of sympathetic to parasympathetic activity was increased by taurine depletion.

Baroreflex control of heart rate was significantly decreased in both taurine-depleted groups (Fig. 13.4), but the decrease was significantly greater in taurine depleted rats maintained on a high glucose diet; a high glucose diet did not affect baroreflex regulation of control rats. Baroreflex control of renal nerve activity (an

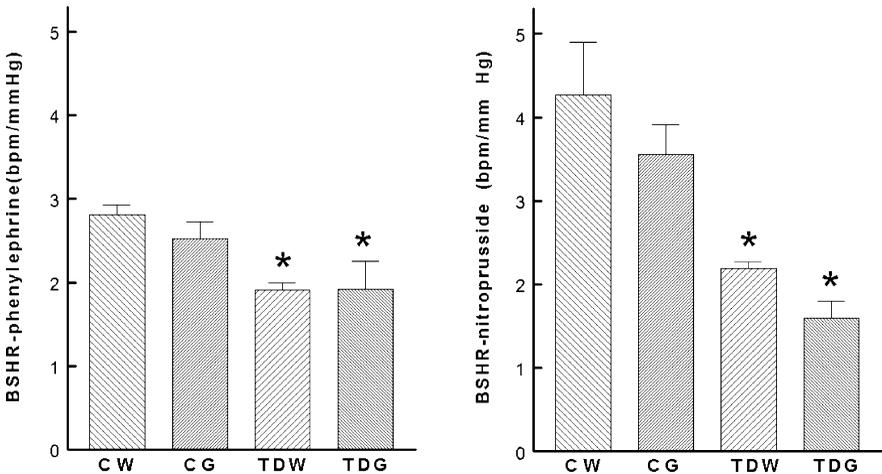


Fig. 13.4 Perinatal taurine depletion decreases the control of baroreflex sensitivity of heart rate in adult, male rats (* $P < 0.05$ to CW). See text for abbreviations

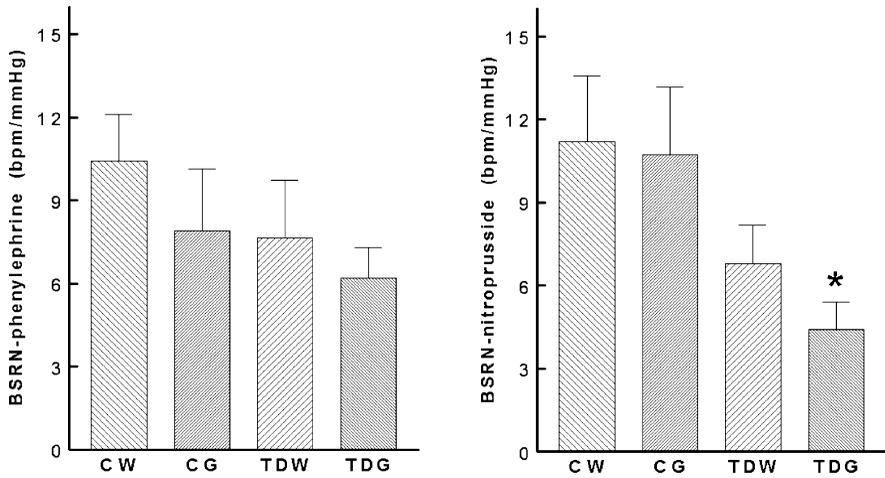


Fig. 13.5 High sugar intake aggravates baroreflex sensitivity control of renal nerve activity in perinatal taurine depleted rats ($*P < 0.05$ to CW). See text for abbreviations

indicator of autonomic nervous system control of renal vascular resistance) was also blunted by perinatal taurine depletion and was further blunted by high dietary glucose in the taurine depleted rats (Fig. 13.5). Further, baroreflex control of arterial pressure (within the normal range of arterial pressure, i.e., 80–120 mm Hg) was consistently blunted by perinatal taurine depletion.

13.4 Discussion

During perinatal life, large alterations in nutritional intake can lead to obesity, insulin resistance, hypertension, and other cardiovascular symptoms. These effects may be initiated during pre- or postnatal development (Barker et al. 2002; Harding 2001; Langley-Evans 2006). The present study indicates that perinatal taurine depletion causes a dysregulation of the autonomic nervous system that is exacerbated by a high sugar diet. Further, these changes in autonomic nervous system regulation may contribute to increased arterial pressure in perinatal taurine-depleted rats on a high glucose diet. This provides another example of how the perinatal environment can alter function in adults and potentially contribute to disease (Barker et al. 2002; Harding 2001; Langley-Evans 2006).

Several lines of evidence indicate that imbalances in maternal protein to carbohydrate intake can result in low birth weight of offspring (Law et al. 2001; Shiell et al. 2001), which subsequently increases the risk of developing insulin resistance and cardiovascular disease. Imbalances in glucocorticoid levels and in the renin-angiotensin system, as well as abnormal nephrogenesis during early life, can modify the adaptive ability of the offspring in later life (Eriksson et al. 2007; Hanson et al. 2004; Mendez et al. 2004). The present study shows that perinatal

taurine depletion in rats slightly decreases body weight, thus supporting the growth-promoting ability of taurine in early life (Lourenco and Camilo 2002). While perinatal taurine depletion could have caused taurine concentrations to remain low throughout life, thereby producing chronic taurine deficiency, this seems unlikely. After weaning, all animals were supplied with normal rat chow that appears to “normalize” taurine content within 4–5 weeks (Pacioretty et al. 2001). However, we did not independently measure taurine content in these rats. In addition, the animals in the present study displayed no signs of insulin resistance or diabetes mellitus. This suggests that insulin resistance may not be the key factor predisposing these animals to subsequent cardiovascular impairment (Reaven 1991). We have previously reported that in this model, high sugar diet can induce renal dysfunction without insulin resistance or hypertension (Roysommuti et al. 2002).

Taurine supplementation during perinatal or adult life reduces hypertension in animal models, including SHR, cyclosporine A-induced hypertension and sugar-induced hypertension (Militante and Lombardini 2002). The present findings indicate that perinatal taurine depletion predisposes animals to dietary sugar-induced hypertension. Lack of taurine in early life may underlie the effect of perinatal protein malnutrition on adult hypertension (Aerts and Van Assche 2002). Interestingly, taurine, which is a sulphur-containing beta-amino acid that is found mainly in animal meat, is not present in most plant proteins and is found in very low levels in cow's milk (Aerts and Van Assche 2002). In contrast, it is in high concentration in human and rat milk. In humans, taurine content is higher in non-vegetarians than vegetarians.

Dietary taurine supplementation appears to be directly related to taurine's ability to decrease sympathetic nerve activity, likely at the level of the central nervous system (Mizushima et al. 1996; Sato et al. 1987). Taurine supplementation also reduces oxidative stress (Aerts and Van Assche 2002; Racasan et al. 2004). Interestingly, all perinatal taurine depleted rats on a normal glucose diet display resting autonomic nerve hypoactivity but sympathetic nerve activity is only selectively increased in rats fed the high sugar diet, suggesting that perinatal taurine depletion may not retard growth and development of the autonomic nervous system, but may dysregulate it. The selective action of high sugar intake on the regulation of the sympathetic nervous system suggests that taurine deficiency in early life may alter the adult central nervous system. Taurine injection into the brain has been shown to decrease sympathetic outflow in animals (Inoue et al. 1985); however, this effect does not appear to be direct, but rather acts through some other mechanism, e.g., adenosine or glutamate receptor systems (Albrecht and Schousboe 2005; Kohlenbach and Schlicker 1990). The hypothalamic sympathetic pathways play a very important role in pathogenesis of many models of hypertension (Carlson et al. 2001). Taurine depletion during development may modify the function of this brain area.

Baroreceptor reflex plays a crucial role in minute-to-minute regulation of arterial pressure, and recent evidence suggests that baroreceptor dysfunction may contribute to hypertension in some animal models, e.g., SHR (Carlson et al. 2001). In sustained hypertension, baroreflex sensitivity is usually blunted, either by arterial vascular wall stiffness or central nervous system adjustments, i.e., baroreflex resetting. In

the present study, baroreflex control of both heart rate and renal nerve activity was blunted in perinatal taurine-depleted rats, even though mean arterial pressure and heart rate were not significantly different in the taurine-depleted rats compared to the control rats. In contrast, glucose supplementation led to both baroreflex blunting and increased arterial pressure in the taurine-depleted but not in the control rats. Together, this suggests that postprandial hyperglycemia and/or hyperinsulinemia may lead to sustained sympathetic overactivity and hypertension due to insufficient baroreceptor reflex adjustments. Renal nerve activity reflects sympathetic nervous system activity but not parasympathetic nervous system activity. Thus, the blunted baroreflex control of renal nerve activity by a high sugar diet in the perinatal taurine-depleted rats indicates that the changes in cardiovascular control in these rats are due primarily to the actions of sympathetic (as opposed to parasympathetic) pathways. However, blunted renal excretory function is likely not to be the primary cause of hypertension in these animals, since natriuretic and diuretic function is not different between the groups (Roysommuti et al. 2004).

The renin-angiotensin system plays an important role in the pathogenesis of arterial hypertension, including sugar-induced hypertension. Our previous study indicates that high sugar intake impairs renal function before the development of insulin resistance and hypertension (Roysommuti et al. 2002). This effect is abolished by treatment with angiotensin converting enzyme inhibitor, captopril. The present experiment also further indicates that high dietary glucose does not alter autonomic nervous system function in control rats. It is possible that a glucose-induced rise in angiotensin II may suppress the central baroreflex pathway (DiBona and Jones 2003; McMullan et al. 2007) and activate sympathetic nerve activity (Gao et al. 2005; Johns 2005) in the perinatal taurine depleted rats, which are more susceptible to pressor agents than the control. Overexpression of angiotensin II receptors has also been reported in perinatal protein restricted offspring (Pladys et al. 2004; Riviere et al. 2005), but not in adult, perinatal taurine depleted animals.

In summary, while perinatal taurine-depletion blunts baroreceptor reflexes and suppresses autonomic nervous system activity, excess dietary glucose increases sympathetic nerve activity. The baroreceptor reflex was also further blunted by high sugar intake. These data further support an important role for maternal dietary taurine during the perinatal period.

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