Birth Weight Is Inversely Correlated to Adult Systolic Blood Pressure and Pulse Pressure in Type 1 Diabetes

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Abstract—In the general population, there is an inverse relationship between birth weight and adult systolic blood pressure. Because blood pressure in diabetic patients at least in part seems to be regulated by different mechanisms than in nondiabetic subjects, it is not known whether a similar correlation exists in diabetic individuals. Therefore, we obtained data on birth weight from original birth certificates in 1543 type 1 diabetic patients. Blood pressure was measured auscultatorily on a single occasion. In the 1225 patients born at term (after 37 weeks of gestation), the age- and sex-adjusted regression coefficients between systolic blood pressure and birth weight was \(-1.90\) mm Hg/kg (95% confidence interval [CI], \(-3.71\) to \(-0.09\)). The finding remained unchanged after adjustment for body mass index, current smoking, duration of diabetes, social class, antihypertensive therapy, glomerular filtration rate, glycemic control, and elevated albuminuria. The regression coefficient between birth weight and pulse pressure was of a similar magnitude. The age-adjusted regression coefficient between systolic blood pressure and birth weight seemed stronger in females (\(-3.34\) mm Hg/kg; 95% CI, \(-6.06\) to \(-0.62\)) than in males (\(-0.42\) mm Hg/kg; 95% CI, \(-2.80\) to \(1.95\)), although this difference was not statistically significant. As a new finding, we report an inverse relationship between weight at birth and systolic blood pressure and pulse pressure in adult type 1 diabetic patients. Given the deleterious effects of elevated arterial blood pressure in diabetes, the impact of intrauterine growth retardation on the development of end-organ damage needs to be clarified. (Hypertension. 2004;44:1-6.)

Key Words: diabetes mellitus ■ blood pressure ■ epidemiology

Small size at birth has been linked to an increased morbidity in adult life. For instance, individuals with low birth weight have been found to be at increased risk for cardiovascular disease, diabetes, renal disease, and elevated blood pressure.\(^1\)\(^-\)\(^4\) It has been postulated that the elevated blood pressure observed in individuals with low birth weight could be a result of the intrauterine environment: malnutrition during the fetal period could cause permanent changes in the organs involved in blood pressure regulation.\(^5\) Alternatively, the relation between low birth weight and blood pressure could result from common underlying genetic factors influencing both parameters.\(^6\) Recent studies in monzygotic and dizygotic twin pairs have reported evidence in support of the latter view.\(^7\)\(^8\)

Elevated blood pressure is a common feature of type 2 diabetes, whereas diabetes-specific blood pressure derangements in type 1 diabetic patients generally have been thought of as isolated to the subgroup of patients with diabetic kidney disease.\(^9\) However, more recently, emphasis has been put on the effects of blood pressure induced by the metabolic alterations associated with type 1 diabetes. In animal models of type 1 diabetes, the onset of hyperglycemia causes an increase in arterial blood pressure.\(^10\) Furthermore, in humans, combined kidney/pancreas transplantsations have been found to cause a dramatic decrease in blood pressure compared with isolated kidney transplantsations, a finding that underscores the importance of the diabetic state in the pathogenesis of hypertension in type 1 diabetes. In support of diabetes-specific effects on blood pressure occurring even in the absence of renal complications in type 1 diabetes, we have recently reported a premature age-induced increase in pulse pressure in type 1 diabetic patients compared with nondiabetic subjects.\(^12\) Therefore, even in the absence of diabetic kidney disease, the mechanisms and patterns of blood pressure derangements seem to differ between subjects with type 1 diabetes when compared with nondiabetic subjects.

Epidemiological studies in the general population have consistently reported an inverse relationship between birth weight and systolic blood pressure later in life.\(^1\)\(^-\)\(^3\) Because the pathogenetic mechanisms of elevated blood pressure may be
different in diabetic subjects, the inverse relationship between birth weight and blood pressure found in the general population cannot be directly extrapolated to diabetic subjects. We are not aware of any previous studies addressing the issue specifically in type 1 diabetic patients; therefore, the aim of the present study was to assess the relationship between birth weight and blood pressure in a large sample of type 1 diabetic patients.

**Methods**

The Finnish Diabetic Nephropathy (FinnDiane) study is a nationwide, prospective, multicenter study that was initiated in November 1997. The patients are recruited from 20 university and central hospitals, from 23 local hospitals, and from 16 primary health care centers. The study protocol is in accordance with the Declaration of Helsinki and it has been approved by the local ethics committee in each participating study center.

The present study presents cross-sectional data from the baseline visit. All patients with a diagnosis of type 1 diabetes (code E10 in International Classification of Diseases, 10th version) attending the diabetic and renal outpatient clinics and dialysis units were consecutively asked to participate. Before participation, all patients gave their written informed consent and the response rate was 78%. Based on medical records, the attending local physician completed a study form assessing diabetic microvascular and macrovascular late complications. The patients were asked to complete a questionnaire dealing with place of birth, smoking habits, alcohol intake, and social class.

The degree of renal involvement was classified in the coordinating center using all available data on albuminuria drawn from local medical records. Diabetic nephropathy was defined as a urinary albumin excretion rate (UAER) exceeding 200 μg/min (overnight collections) or 300 μg/24 hours (24-hour urine collections), or as a urinary albumin/creatinine ratio in a spot sample exceeding 35 mg/mmol (male) or 25 mg/mmol (female), in 2 out of 3 consecutive measurements. The corresponding cutoff values for microalbuminuria were 20 to 200 μg/min or 30 to 300 μg/24 hours for UAER, and 3.5 to 35 mg/mmol (male) or 2.5 to 25 mg/mmol (female) for urinary albumin/creatinine ratio. Two UAER measurements within the normal range were required for the classification of a patient as normoalbuminuric. The degree of renal involvement was considered to be unclassifiable if there were insufficient data on UAER, or if elevated UAER of an origin other than diabetes (pregnancy, nondiabetic kidney disease, duration of diabetes <3 years) was clinically suspected. The classification was based on historic data in cases in which UAER had decreased as a result of intervention (antihypertensive therapy).

For this study, all 3115 patients with an age at onset of diabetes younger than 36 years, who had had insulin therapy initiated within 1 year after diagnosis, and who were studied by October 31, 2002 were selected. Of the 2313 patients who reported birth at a hospital, we were able to identify the birth hospital of 1956 patients. After exclusion of 23 twin pregnancies, the original birth certificate with data on birth weight was obtained for 1543 patients. Of these, 1506 had data on birth height, whereas 1433 had data on gestational age (calculated from the last menstruation before pregnancy).

**Measurements**

Blood pressure measurements were performed by a trained nurse at the outpatient clinic. The measurement was performed auscultatorily on the right arm with a calibrated mercury sphygmomanometer with the subject in sitting position after 5 to 10 minutes of rest. Systolic (Korotkoff I) and diastolic blood pressures (Korotkoff V) were recorded to the closest 2 mm Hg, and the mean value of 2 recordings was used in the analysis.

**Laboratory Assays**

Serum creatinine concentration (normal reference values: male <115 μmol/L, female <100 μmol/L) was measured with a kinetic Jaffé reaction. Glomerular filtration rate (GFR) was calculated with the Cockcroft–Gault formula ((140−age [years])×weight [kg])/ [0.815×serum creatinine (μmol/L)]×0.85 in women), both corrected for body surface area. Data on HbA1c, and albuminuria were drawn from local medical records.

**Statistics**

All analyses were performed using the SPSS 11.5 statistical package. The significance of differences in categorical variables was assessed with the χ² test, whereas continuous variables were analyzed with ANOVA (normally distributed) or the Mann–Whitney test (non-normally distributed). The correlation between birth weight and blood pressure variables was assessed with linear regression analysis. The effect of potential confounding factors was assessed by entering them into a statistical model one by one. Because of previous results reporting evidence for a sex-specific effect of birth weight in type 1 diabetes, and to control for the potentially confounding effect of gestational age, stratified analyses were performed in predefined subgroups (males versus females; preterm versus full-term delivery). The significance of a sex-specific effect on blood pressure was formally tested by adding an interaction term (birth weight multiplied by sex) to the regression model consisting of birth weight, age, and sex. P<0.05 was considered statistically significant.

**Results**

The clinical characteristics of the studied patients are presented in Table 1. Of the 3115 type 1 diabetic patients, data on birth weight were available in half of the patients. As expected, birth data were more readily available in younger patients. In addition, compared with patients without data on birth weight, a higher frequency of the studied patients were females, they had shorter duration of diabetes, and had less microvascular and macrovascular complications. The patients studied also had lower systolic blood pressure, whereas no difference was found in diastolic blood pressure.

In a linear regression analysis adjusted for current age and sex, the regression coefficient between systolic blood pressure and birth weight was −0.96 mm Hg/kg (95% confidence interval [CI] −2.54 to 0.63). This coefficient remained largely unchanged after further adjustments for body mass index (BMI), current smoking, duration of diabetes, social class, antihypertensive therapy, glomerular filtration rate, glycemic control, and elevated urinary albumin excretion rate. The slope between birth weight and pulse pressure was of similar magnitude (data not shown).

When the analysis was restricted to the 1225 patients born at term (after 37 weeks of gestation), the age- and sex-adjusted regression coefficients between systolic blood pressure and birth weight was −1.90 mm Hg/kg (95% CI, −3.71 to −0.09). Further adjustments for the aforementioned potential confounding factors strengthened this correlation slightly (regression coefficient −2.37 mm Hg/kg; 95% CI, −4.08 to −0.65). The patients born at term were comparable to patients born preterm regarding current age, duration of diabetes, gender distribution, glycemic control, BMI, blood pressure levels, smoking habits, and social class (data not shown).

The regression coefficients between birth weight and adult blood pressure variables varied substantially between females and males (Tables 2 and 3). In females, significant negative correlations between birth weight and systolic blood pressure and pulse pressure were found. Birth weight was also nega-
Table 1. Characteristics of the Studied Patients in Comparison With Patients Without Data on Birth Weight

<table>
<thead>
<tr>
<th>Variable</th>
<th>Birth Weight Data Available</th>
<th>Birth Weight Data Not Available</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>1543</td>
<td>1572</td>
<td></td>
</tr>
<tr>
<td>Males, %</td>
<td>49</td>
<td>55</td>
<td>0.001</td>
</tr>
<tr>
<td>Age, y</td>
<td>32.8±9.6</td>
<td>42.3±11.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age at onset, y</td>
<td>13.9±7.9</td>
<td>16.4±8.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes duration, y</td>
<td>19.0±10.6</td>
<td>25.9±11.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height, cm</td>
<td>170.7±9.3</td>
<td>170.6±9.4</td>
<td>NS</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.9±3.5</td>
<td>25.1±3.5</td>
<td>NS</td>
</tr>
<tr>
<td>Current smokers, %</td>
<td>25</td>
<td>23</td>
<td>NS</td>
</tr>
<tr>
<td>Antihypertensive therapy, %</td>
<td>34</td>
<td>50</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Blue collar workers, %</td>
<td>53</td>
<td>63</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HbA1c, %</td>
<td>8.5±1.6</td>
<td>8.5±1.5</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>131±17</td>
<td>138±20</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>80±10</td>
<td>80±10</td>
<td>NS</td>
</tr>
<tr>
<td>Mean arterial blood pressure, mm Hg</td>
<td>97±10</td>
<td>99±10</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse pressure, mm Hg</td>
<td>51±14</td>
<td>58±17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Elevated albuminuria, %</td>
<td>32</td>
<td>41</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>End-stage renal disease, %</td>
<td>5</td>
<td>12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Glomerular filtration rate, ml/min per 1.73 m²*</td>
<td>95±32</td>
<td>87±31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>History of laser treatment, %</td>
<td>31</td>
<td>47</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>History of macrovascular disease, %</td>
<td>5</td>
<td>14</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Birth weight, g</td>
<td>3512±526</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Birth height, cm</td>
<td>50.4±2.1</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Gestational age, d</td>
<td>277±14</td>
<td>NA</td>
<td></td>
</tr>
</tbody>
</table>

Data are proportion or mean±SD.
NS indicates not significant; NA, not assessed.
*Patients with end-stage renal disease excluded (n=269).

Table 2. Regression Coefficients Between Birth Weight and Blood Pressure in Females

The data presented are based on females with complete data on all variables (52 females with missing data were excluded).
BP indicates blood pressure.
*P<0.05; †P<0.01; ‡P<0.001.
pressure or pulse pressure) used as the dependent variable, the interaction term of birth weight and sex did not reach statistical significance ($P=0.14$ for both comparisons).

As depicted in the Figure, the age- and sex-adjusted regression coefficient between birth weight and systolic blood pressure was negative and of similar magnitude in patients with normoalbuminuria, microalbuminuria, overt diabetic nephropathy, and end-stage renal disease. However, the regression coefficient failed to reach statistical significance in the patient groups with elevated UAER. The regression coefficient was $-2.77$ mm Hg/kg ($P=0.005$) in patients with normal renal function ($\text{GFR} > 90$ mL/min per 1.73 m$^2$) and $-2.40$ ($P=\text{NS}$) in patients with mild renal dysfunction ($\text{GFR} 60$ to 90 mL/min per 1.73 m$^2$), whereas it was $+2.26$ mm Hg/kg ($P=\text{NS}$) in patients with moderate to severe renal dysfunction ($\text{GFR} < 60$ mL/min per 1.73 m$^2$).

One-third of the studied patients received blood pressure-lowering medication. The correlation between birth weight and systolic blood pressure was slightly more pronounced in those without antihypertensive therapy (regression coefficient $-1.52$; 95% CI, $-3.06$ to 0.26 mm Hg/kg; $P=0.054$) compared with those receiving treatment for hypertension ($-0.04$; 95% CI, $-3.00$ to 2.92 mm Hg/kg; $P=\text{NS}$). There was no statistically significant differences between different types of antihypertensive drugs (angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, $\beta$-blockers, calcium channel blockers or diuretics; data not shown).

### Discussion

As a new finding, we report an inverse relationship between birth weight and both systolic blood pressure and pulse pressure in patients with type 1 diabetes.

In the general population, a 1-kg decrease in birth weight has been found to increase systolic blood pressure by 2 to 4 mm Hg. The clinical importance of this finding remains to be elucidated. To our knowledge, the present study is the first to address the relation between factors operating in utero and adult blood pressure specifically in individuals exposed to type 1 diabetes. This is an important difference, because individuals with diabetes are especially susceptible to blood pressure-induced end-organ damage. Consequently, even a small effect of birth weight on blood pressure may be clinically relevant.

We have recently reported an earlier age-related increase in systolic blood pressure and an earlier decrease in diastolic blood pressure resulting in a premature increase in pulse pressure in type 1 diabetic patients when compared with the nondiabetic background population. Most interestingly, this premature increase in pulse pressure was evident even in patients without any signs of diabetic kidney disease. Similarly, several other lines of evidence have underlined the importance of metabolic factors in the pathogenesis of hypertension in type 1 diabetes. Despite potential differences in the pathogenesis of elevated blood pressure, birth weight seems to have a similar effect on adult blood pressure in type 1 diabetes as in the general population. Interestingly, in contrast with our findings, a recent study in Taiwanese type 2 diabetic school children revealed that those with a high

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**TABLE 3. Regression Coefficients Between Birth Weight and Blood Pressure in Males**

<table>
<thead>
<tr>
<th>Regression Coefficients Adjusted for</th>
<th>All Males (n=703)</th>
<th>Males Born at Term (n=591)</th>
<th>All Males (n=703)</th>
<th>Males Born at Term (n=591)</th>
<th>All Males (n=703)</th>
<th>Males Born at Term (n=591)</th>
<th>All Males (n=703)</th>
<th>Males Born at Term (n=591)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Age</td>
<td>$-0.14$</td>
<td>$-0.42$</td>
<td>$0.36$</td>
<td>$0.38$</td>
<td>$0.20$</td>
<td>$0.12$</td>
<td>$-0.50$</td>
<td>$-0.81$</td>
</tr>
<tr>
<td>Model 2: Model 1 + BMI</td>
<td>$-0.21$</td>
<td>$-0.48$</td>
<td>$0.27$</td>
<td>$0.30$</td>
<td>$0.11$</td>
<td>$0.04$</td>
<td>$-0.48$</td>
<td>$-0.79$</td>
</tr>
<tr>
<td>Model 3: Model 2 + current smoking</td>
<td>$-0.21$</td>
<td>$-0.49$</td>
<td>$0.27$</td>
<td>$0.30$</td>
<td>$0.11$</td>
<td>$0.04$</td>
<td>$-0.44$</td>
<td>$-0.79$</td>
</tr>
<tr>
<td>Model 4: Model 3 + duration of diabetes</td>
<td>$-0.17$</td>
<td>$-0.56$</td>
<td>$0.27$</td>
<td>$0.31$</td>
<td>$0.12$</td>
<td>$0.02$</td>
<td>$-0.41$</td>
<td>$-0.87$</td>
</tr>
<tr>
<td>Model 5: Model 4 + social class</td>
<td>$-0.14$</td>
<td>$-0.51$</td>
<td>$0.26$</td>
<td>$0.31$</td>
<td>$0.20$</td>
<td>$0.04$</td>
<td>$-0.41$</td>
<td>$-0.82$</td>
</tr>
<tr>
<td>Model 6: Model 5 + antihypertensive therapy</td>
<td>$-0.17$</td>
<td>$-0.56$</td>
<td>$0.24$</td>
<td>$0.28$</td>
<td>$0.18$</td>
<td>$0.00$</td>
<td>$-0.38$</td>
<td>$-0.83$</td>
</tr>
<tr>
<td>Model 7: Model 6 + GFR</td>
<td>$-0.13$</td>
<td>$-0.56$</td>
<td>$0.25$</td>
<td>$0.27$</td>
<td>$0.12$</td>
<td>$0.00$</td>
<td>$-0.38$</td>
<td>$-0.82$</td>
</tr>
<tr>
<td>Model 8: Model 7 + HbA1c</td>
<td>$-0.13$</td>
<td>$-0.54$</td>
<td>$0.24$</td>
<td>$0.31$</td>
<td>$0.12$</td>
<td>$0.02$</td>
<td>$-0.38$</td>
<td>$-0.85$</td>
</tr>
<tr>
<td>Model 9: Model 8 + elevated albuminuria</td>
<td>$-0.05$</td>
<td>$-0.53$</td>
<td>$0.19$</td>
<td>$0.11$</td>
<td>$0.23$</td>
<td>$-0.27$</td>
<td>$-0.34$</td>
<td>$-0.84$</td>
</tr>
<tr>
<td>Model 10: Model 9 + height</td>
<td>$-0.35$</td>
<td>$-1.02$</td>
<td>$0.19$</td>
<td>$0.11$</td>
<td>$0.23$</td>
<td>$-0.27$</td>
<td>$-0.54$</td>
<td>$-1.13$</td>
</tr>
</tbody>
</table>

The data presented are based on males with complete data on all variables (56 males with missing data were excluded). None of the regression coefficients reached statistical significance.
Birth weight were those who had the highest blood pressure at the age of 6 to 18 years. We are not aware of any other studies assessing the birth weight–blood pressure relationship specifically in diabetic individuals. However, based on these diverging results in type 1 and type 2 diabetes, the impact of size at birth on blood pressure later in life may be modulated by factors associated with the diabetic state itself.

In type 1 diabetic subjects susceptible to diabetic kidney disease, blood pressure increases as renal injury progresses. However, although the regression coefficient was not significant within the subgroups, the inverse relationship between birth weight and blood pressure seems to persist despite considerable evidence of renal injury, either based on elevated albuminuria or on diminished glomerular filtration rate. However, the inverse relationship was no longer evident in patients with moderate to severe renal dysfunction.

In line with findings in the general population, birth weight was related to systolic, but not to diastolic, blood pressure. Sympathetic overactivity is thought to particularly influence systolic blood pressure via mechanisms such as increased cardiac output, reduced arterial compliance, and increased arteriolar vasoconstriction, resulting in an enhanced reflection of the arterial wave from the periphery. A recent study in nondiabetic twins found that the inverse relationship between birth weight and blood pressure to a large extent was explained by increased sympathetic activity in patients with low birth weight. Although not studied in diabetic patients, effects on the sympathetic nervous system could offer an explanation for the stronger effect of birth weight on the pulsatile component of blood pressure.

A sex-stratified analysis revealed that the inverse correlation between birth weight and systolic blood pressure was stronger in females, although the difference between females and males did not reach statistical significance. Because intrauterine growth retardation has been suggested to predispose not only to systemic hypertension but also to intraglomerular hypertension and progressive renal disease, it is interesting that in the only study published thus far that deals with the impact of low birth weight on development of diabetic kidney disease in type 1 diabetes, the finding was isolated to female patients. In the general population, although occasionally reported, no overall sex-specific difference in the birth weight–blood pressure relationship seems to exist. Although the impact of size at birth on health in later life may be stronger in female than in male type 1 diabetic patients, this is still a mere hypothesis that urges further testing before further conclusions can be drawn.

The impact of birth weight on adult blood pressure was more pronounced in patients born at term. This could imply that inappropriate intrauterine growth for a certain gestational age, rather than the actual birth weight, is a stronger determinant of blood pressure in adulthood. Alternatively, the last weeks of a pregnancy of normal duration may be required for some pathological mechanisms to emerge. Recent results speak in favor of the latter explanation as intrauterine growth retardation was associated with endothelial dysfunction at 3 months of postnatal age only in children born at term, but not in children born preterm. Beyond doubt, our data demonstrate that gestational age is a central variable in studies assessing the impact of size at birth on blood pressure later in life.

Our study has some limitations. We are aware that the cross-sectional study design is susceptible to selection bias. We may therefore have missed patients born small and, according to the hypothesis, at higher risk for cardiovascular morbidity and mortality. However, this would lead to an underestimation rather than overestimation of the birth weight–blood pressure relationship. Also, we did not have birth data on the whole cohort, because the data were often not available in older patients, for instance, those born before the 1950s. Because the patients with birth data available were younger, this may have contributed to the fact that they had lower blood pressure, less antihypertensive medication, and less microvascular and macrovascular complications. In the older patients without birth data, especially those born during and after World War II, birth weight was probably lower than in the studied patients. Because both systolic blood pressure and pulse pressure were higher in the proportion of patients without birth data, it seems plausible to assume that the actual impact of birth weight on blood pressure could be even stronger than what we were able to observe.

We measured the blood pressure on one occasion, and the use of repeated measurements or more sophisticated methods would have resulted in a more precise estimation of the actual blood pressure levels. Our methodology, however, is more robust than the one used in many large-scale studies in the general population, in which patient-reported blood pressure levels have been used. One additional strength of our study is that we had the opportunity to extract birth data directly from original birth certificates instead of using self-reported data.

**Perspectives**

Birth weight is inversely related to systolic blood pressure and pulse pressure in adulthood in patients with type 1 diabetes. This association is especially pronounced in female patients, in whom a 1-kg lower birth weight is associated with a 3- to 4-mm Hg higher systolic blood pressure. Given the deleterious effects of elevated arterial blood pressure in diabetes, the impact of intrauterine growth retardation on the development and progression of end-organ damage in diabetic subjects needs to be further evaluated.

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