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BRIEF REPORT

Birth weight predicts both proteinuria and overweight/obesity in a rural population of Aboriginal and non-Aboriginal Canadians

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The risk for many chronic diseases appears to be mediated in part by birth weight. Among Aboriginal Canadians, the prevalence of end-stage renal disease and cardiovascular disease risk is disproportionately high, largely because of elevated diabetes prevalence. The relationships between birth weight (and other potential risk factors) and diabetes, hypertension, proteinuria and overweight/obesity were explored in 1439 rural Albertans (Canada), of whom 67.3% were Aboriginal. At voluntary outreach screening programs, demographic and clinical data were measured and recalled birth weights recorded. Statistical modeling using logistic regression was used to evaluate the relationships. In the final adjusted models, associations remained for low birth weight and proteinuria [odds ratio (OR) 2.36; 95% CI 1.24–4.49], as well as for high birth weight and overweight/obesity (OR 1.58; 95% CI 1.00–2.53). These findings emphasize the need to strive for healthy pregnancies, with appropriate weight gains in these and other disadvantaged populations around the world.

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Key words: aboriginal health, birth weight, programming, renal, type 2 diabetes

Introduction

Low birth weight (LBW) was first proposed to be a risk factor for adult cardiovascular disease in 1986.[1] Studies conducted around the world have confirmed associations between LBW and subsequent above average body mass with type 2 diabetes, hypertension, cardiovascular disease and stroke. Recently, a significantly increased risk for chronic kidney disease (CKD) among LBW infants in various populations was shown through systematic review.[2] Moreover, a U-shaped relationship with birth weight and both increased urine albumin excretion and end-stage renal disease was shown among Pima Indians and individuals from the southern United States, especially among subjects with type 2 diabetes.[3,4] Similar U-shaped associations have been found between birth weight and type 2 diabetes in Indigenous populations.[5,6]

CKD is disproportionately more prevalent among Aboriginal Canadians (First Nations, Métis and Inuit) compared with other Canadian populations.[7] For instance, advanced CKD (glomerular filtration rate <30 ml/min per 1.7 m^2) was nearly twofold higher among First Nations (5.9/1000) compared with non-First Nations (3.8/1000) in Alberta.[8] Similarly, the incidence and prevalence of type 2 diabetes is at least two times higher among First Nations compared with the general population in Canada.[9] Although high birth weight (HBW) was associated with increased prevalence of type 2 diabetes and end-stage renal disease among Aboriginal people in Saskatchewan,[6] we know little of the relationship between birth weight and established risk factors for CKD among other rural or Aboriginal Canadian populations. Our objective in this study was to investigate the associations between birth weight and known risk factors for cardiovascular and renal diseases: overweight/obesity, type 2 diabetes, hypertension and proteinuria.

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Method

Canadians from Aboriginal communities or rural towns in northern Alberta took part in voluntary programs screening for diabetes and its complications as part of two separate projects. Individuals gave consent for aggregate analysis of their data, and both projects were approved by the University of Alberta Health Research Ethics Board. Since 2001, the Screening for Limb, I-Eye, Cardiovascular and Kidney complications of diabetes (SLICK) project has travelled to each of the 44 Alberta First Nations communities providing diabetes risk assessment, diabetes complications screening and community-based care.[10] The Mobile Diabetes Screening Initiative (MDSi) has travelled to rural Aboriginal 'off-reserve' and remote Alberta communities providing diabetes screening since 2003.[11] Both projects deploy mobile clinics equipped with portable technology and health-care personnel into the communities. All subjects were counseled on-site regarding testing results and encouraged to follow up with their physicians. The data presented herein were collected between July 2002 and December 2011.

Participants self-referred in response to local advertising and underwent visits with trained health-care workers. Measured data included waist circumference, body mass index (BMI), blood pressure, blood glucose (fasting or random), hemoglobin A1c (A1c), total cholesterol, high-density lipoprotein (HDL), random dipstick urine protein and random urine albumin to creatinine ratio (ACR). Subjects removed their shoes and were dressed in light clothing to be weighed, and waist circumference was measured using a standard measuring tape at the iliac crest. Subjects rested for 5 min before a single seated blood pressure reading. Blood samples were collected via a single finger puncture with the Accu-Chek Safe-T-Pro (Roche Diagnostics) lancet after hand washing and finger sanitizing. A1c and ACR values were determined using the Bayer DCA2000® + analyzer. Lipids were detected using the Cholestech L.D.X™ portable analyzer. Self-reported data included age, sex, ethnicity, medication usage, birth weight, activity level (whether they engaged in little or no physical activity in most weeks), prematurity (born at <37 weeks' gestation), type 2 diabetes history and gestational diabetes mellitus (GDM) history.

Waist circumference was considered high if >102 cm in men, >88 cm in women or >90th percentile in children.[12,13] Criteria from the National Cholesterol Education Program Adult Treatment Panel III[12] were utilized to define overweight (BMI 25–29.9 kg/m^2) and obesity (BMI ⩾ 30 kg/m^2) in adults. Among children, BMI between the 85th and 95th percentile determined overweight and ⩾95th percentile indicated obesity.[14] Hypertension was defined as a blood pressure above the 90th percentile in children;[15] in adults it was defined as a systolic or diastolic blood pressure >140/90 mmHg for those without type 2 diabetes, >130/80 mmHg for those with type 2 diabetes[16] and/or the use of antihypertensive medication. Type 2 diabetes was defined as a positive documented history of type 2 diabetes and/or use of insulin or hypoglycemic medication; it was also defined as an A1c ⩾6.5%, a fasting plasma glucose level ⩾7.0 mmol/l or a random plasma glucose level ⩾11.0 mmol/l.[16,17] We excluded those with type 1 diabetes on the basis of clinical assessment of age at diagnosis, history of diabetic ketoacidosis, weight, family history and dose of insulin. Total cholesterol to HDL ratio ⩾4.0 was considered high, irrespective of treatment.[16] Dipstick-positive proteinuria and/or an ACR > 2.74 in females or >1.94 in males was used to identify proteinuria.[16] We classified birth weight as low (LBW ⩽ 2500 g), normal (NBW 2501–3999 g) and high (HBW ⩾ 4000 g).[18] Reliable data on gestational age were not available.

Subjects self-reported their ethnicity as First Nations, Métis or non-Aboriginal. Individuals who identify as Indian (First Nations), Métis or Inuit are recognized in the 1982 Canadian Constitution Act. Members of First Nations in Canada generally have a legal Indian Status (Registered Indians) according to the Indian Act of Canada. A 'non-Status' Indian is a person who considers himself or herself Indian or a member of a First Nation but who is not registered for a variety of reasons. We included both these groups as 'First Nations'. The Métis is a group with a distinct culture descending from mixed First Nations and European ancestry. Non-Aboriginal subjects in this cohort were Caucasian, largely from Mennonite communities. According to 2006 census data, there were 97 275 First Nations individuals and 85 500 Métis individuals residing in Alberta, corresponding to 3.0% and 2.6% of the Alberta population, respectively.[19]

Statistics

Descriptive analyses were stratified by self-identified ethnicity and sex. Group comparisons were evaluated using univariate analysis of variance for continuous variables and χ^2 tests for categorical variables; all tests of statistical significance were two sided. Statistical modeling (purposeful) using logistic regression was used to evaluate the relationships between birth weight (and other explanatory variables) and diabetes, hypertension, proteinuria and overweight/obesity. All statistical analyses were performed using STATA statistical software (version 11, StataCorp).

Results

A total of 1439 subjects with a mean age of 32.1 ± 17.2 years (range 5–90 years) were included in this analysis. Twenty-nine percent of subjects were under the age of 20 years, 65% between the ages of 20 and 59 years and 6% over the age of 60 years. Ethnicity was documented in 1433 subjects, of whom 469 (32.7%) were non-Aboriginal, 387 (27.0%) were First Nations and 577 (40.3%) were Métis. The majority of subjects (67.4%) were female. Mean birth weight was 3333 ± 772 g (range 454–6747 g). LBW was present in 185 subjects (12.9%), NBW in 1009 (70.1%) and HBW in 245 (17.0%). A total of 156 individuals (12.3%) self-reported as being premature at birth. Type 2 diabetes was present in 134 subjects (9.3%), hypertension in 327 (23.4%) and a high total cholesterol to HDL ratio in 464 (38.7%). The majority of individuals had a high waist circumference (63.3%) and were either overweight or obese (76.8%). The self-reported physical inactivity rate was 41.6%. Among women (⩾20 years of age), 59 (7.8%) ever reported having GDM. Of the 374 subjects who underwent urine testing, 21.7% were classified as having proteinuria. Of them, 30% had diabetes (n = 24/81). Descriptive statistics and group differences are presented in Tables 1 and 2.

Type 2 diabetes (P = 0.011), proteinuria (P = 0.018) and overweight/obesity (P = 0.004) were associated with birth weight in univariate analyses. Significant differences in the prevalence of type 2 diabetes, proteinuria and overweight/obesity were observed across birth weight categories (Fig. 1). An inverse association between birth weight and both type 2 diabetes and proteinuria was present. Interestingly, a U-shaped association between birth weight and overweight/obesity was observed (Fig. 1).

Table 1. *Descriptive analyses by self-identified Aboriginal status*

Variable	Non-Aboriginal ($n = 469$)	First Nations ($n = 387$)	Métis ($n = 577$)	*P*-value
Male	31.3 (27.2–35.8)	34.1 (29.4–39.1)	32.9 (29.1–36.9)	0.686
Birth weight				
LBW	14.1 (11.1–17.6)	10.3 (7.5–13.8)	13.3 (10.7–16.4)	0.246
NBW	70.4 (66.0–74.5)	71.6 (66.8–76.0)	69.2 (65.2–72.9)	0.754
HBW	15.6 (12.4–19.2)	18.1 (14.4–22.3)	17.5 (14.5–20.9)	0.574
Birth weight (g)	3334.9 (782.6)	3337.6 (723.6)	3331.7 (794.7)	0.993
Premature	9.6 (7.1–12.7)	16.8 (13.0–21.3)	11.7 (9.0–14.9)	0.009
Age (years)	39.6 (18.1)	25.4 (14.1)	30.3 (16.1)	<0.001
BMI				
Normal	26.1 (22.1–30.4)	24.7 (20.5–29.4)	20.0 (16.8–23.5)	0.052
Overweight	28.3 (24.2–32.6)	29.3 (24.7–34.1)	24.2 (20.7–27.9)	0.166
Obese	45.7 (41.0–50.3)	46.0 (40.9–51.2)	55.8 (51.6–59.9)	0.001
High WC	54.5 (49.8–59.2)	64.8 (59.4–69.9)	69.6 (65.5–73.6)	<0.001
Inactive	38.5 (33.9–43.2)	34.4 (28.9–40.1)	47.7 (43.5–51.9)	<0.001
Type 2 diabetes	11.1 (8.4–14.3)	5.7 (3.6–8.5)	10.4 (8.0–13.2)	0.014
GDM, adult females only	7.0 (4.3–10.6)	10.7 (6.5–16.4)	7.2 (4.5–10.7)	0.303
Hypertension	27.2 (23.2–31.6)	16.7 (13.0–20.9)	24.8 (21.3–28.6)	0.009
High TC/HDL ratio	36.5 (31.9–41.1)	35.5 (29.5–41.8)	42.2 (37.9–46.7)	0.095
Proteinuria	20.1 (14.3–27.1)	24.3 (14.8–36.0)	22.3 (15.7–30.1)	0.761

LBW, low birth weight; NBW, normal birth weight; HBW, high birth weight; WC, waist circumference; GDM, gestational diabetes mellitus; TC/HDL, total cholesterol/high-density lipoprotein.

Values are percent (95% CI) or mean (S.D) as appropriate.

Table 2. *Descriptive analyses by age group*

Variable	Youth (<20 years; $n = 395$)	Middle-aged (20–59 years; $n = 949$)	Elderly (>59 years; $n = 95$)	*P*-value
Male	49.4 (44.3–54.4)	25.6 (22.9–28.5)	32.6 (23.4–43.0)	<0.001
Birth weight				
LBW	5.8 (3.7–8.6)	14.8 (12.6–17.2)	23.2 (15.1–32.9)	<0.001
NBW	77.5 (73.0–81.5)	68.4 (65.3–71.3)	56.8 (46.3–67.0)	<0.001
HBW	16.7 (13.2–20.8)	16.9 (14.5–19.4)	20.0 (12.5–30.0)	0.726
Birth weight (g)	3334.7 (626.1)	3289.0 (793.0)	3292.5 (1030.9)	<0.001
Premature	14.2 (10.8–18.3)	11.8 (9.7–14.2)	8.0 (3.0–16.6)	0.250
BMI				
Normal	35.1 (30.3–40.1)	19.4 (16.9–22.1)	11.8 (6.1–20.2)	0.052
Overweight	19.4 (15.5–23.7)	30.2 (27.3–33.3)	24.7 (16.4–34.8)	0.166
Obese	45.5 (40.5–50.7)	50.4 (47.1–53.6)	63.4 (52.8–73.2)	0.01
High WC	42.4 (36.5–48.4)	67.9 (64.8–70.9)	80.6 (71.1–88.1)	<0.001
Inactive	22.3 (18.0–27.0)	48.1 (44.7–51.4)	60.0 (45.2–73.9)	<0.001
Type 2 diabetes	0.8 (0.2–2.2)	9.9 (8.1–12.0)	44.2 (34.0–54.8)	<0.001
GDM, adult females only	na	7.8 (5.9–10.1)	7.9 (2.6–17.6)	0.975
Hypertension	30.2 (25.6–35.1)	17.3 (14.9–19.9)	58.2 (47.4–68.5)	<0.001
High TC/HDL ratio	23.6 (18.4–29.4)	42.1 (38.8–45.5)	46.6 (35.9–57.5)	<0.001
Proteinuria	0	21.5 (17.1–26.5)	28.9 (16.4–44.3)	0.083

LBW, low birth weight; NBW, normal birth weight; HBW, high birth weight; WC, waist circumference; GDM, gestational diabetes mellitus; na, not applicable; TC/HDL, total cholesterol/high-density lipoprotein.

Values are percent (95% CI) or mean (S.D.) as appropriate.

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The final adjusted logistic regression models are presented in Table 3. Birth weight was not predictive of type 2 diabetes or hypertension. Significant associations with diabetes were present for age, high total cholesterol HDL ratio, GDM, Métis ethnicity and hypertension. First Nations ethnicity, male sex, high waist circumference and type 2 diabetes were significantly associated with hypertension. Birth weight was associated with proteinuria and overweight/obesity in the respective final models. Hypertension and LBW were predictive of proteinuria. Male sex, age, HBW, Métis ethnicity, hypertension and high total cholesterol HDL ratio were predictive of overweight/obesity.

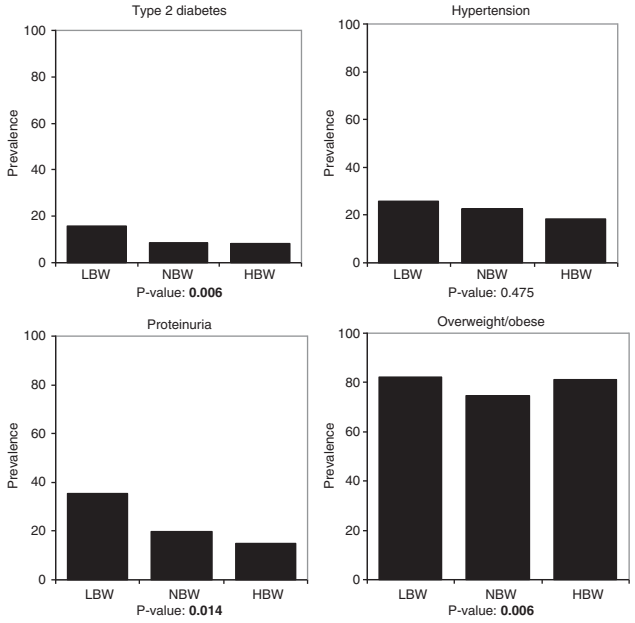

Fig. 1. Prevalence of diabetes, hypertension, proteinuria and overweight/obesity by birth weight category. LBW, low birth weight; NBW, normal birth weight; HBW, high birth weight.

Discussion

Proteinuria and being overweight/obese are well-established risk factors for renal and cardiovascular diseases, which are more common among Aboriginal populations compared with the general Canadian population.[6,9] To our knowledge, this is the first study to show an association between birth weight and proteinuria and overweight/obesity among rural Aboriginal and non-Aboriginal Canadians, which persisted after adjusting for age, gender and other confounding risk factors. Being of LBW conferred a greater than twofold increased risk for proteinuria compared with NBW after controlling for hypertension and type 2 diabetes, demonstrating the independent association with birth weight. This finding is consistent with an odds ratio of 1.8 (1.19–2.77) for proteinuria with LBW, reported in a meta-analysis on 18 studies from around the world.[2] The risk of being overweight/obese in our cohort was 58% higher for those with HBW compared with those with NBW. This is consistent with prior research showing an association of HBW with increased risk of obesity.[5] Proteinuria and obesity are well-recognized risk factors for progression of CKD and cardiovascular disease, especially in young subjects; therefore, our findings have high clinical relevance.[20,21]

Table 3. *Final logistic regression models*

	Type 2 diabetes[a]	Hypertension[b]	Proteinuria[c]	Overweight/obesity[c]
Aboriginal status – First Nations *v.* non-Aboriginal	2.12 (0.87–5.20)	0.47 (0.28–0.77)	na	1.35 (0.87–2.10)
Aboriginal status – Métis *v.* non-Aboriginal	3.34 (1.71–6.53)	0.85 (0.59–1.23)	na	1.70 (1.16–2.49)
Age	1.07 (1.05–1.09)	na	na	1.02 (1.01–1.03)
Birth weight – LBW *v.* NBW	na	na	2.36 (1.24–4.49)	1.40 (0.75–2.61)
Birth weight – HBW *v.* NBW	na	na	0.56 (0.23–1.38)	1.58 (1.00–2.53)
Type 2 diabetes	na	6.98 (4.26–1143)	na	na
GDM (adult females only)	2.67 (1.09–6.56)[d]	na	na	na
Hypertension	6.13 (3.38–11.09)	na	2.25 (1.18–4.28)	2.17 (1.34–3.49)
Inactivity	na	0.94 (0.68–1.30)[d]	na	1.22 (0.86–1.74)[d]
Premature	na	na	na	0.83 (0.46–1.51)[d]
Sex – male *v.* female	na	2.04 (1.40–2.98)	na	0.68 (0.46–0.99)
TC/HDL ratio	2.32 (1.31–4.11)	0.93 (0.84–1.04)[d]	1.18 (0.99–1.39)[d]	2.63 (2.15–3.21)
WC – high *v.* normal	na	2.80 (1.86–4.23)	na	na

na, not applicable; LBW, low birth weight; NBW, normal birth weight; HBW, high birth weight; WC, waist circumference; GDM, gestational diabetes mellitus; TC/HDL, total cholesterol/high-density lipoprotein.

[a] Adjusted for sex.

[b] Adjusted for age.

[c] Adjusted for age and sex.

[d] Included as a confounder.

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We found no associations of birth weight with either type 2 diabetes or hypertension in the full models. This was a surprising finding, as type 2 diabetes and hypertension have been significantly associated with birth weight in many other populations globally.[5,22] However, most studies report higher blood pressure levels as a continuous variable, associated with LBW, which may not become overt hypertension until more advanced age. In our cohort, inclusion of only those with a diagnosis of hypertension likely reduced our ability to detect an association given the relative young age of the cohort. A U-shaped association between birth weight and type 2 diabetes, that is, both LBW and HBW, has been described among some Indigenous populations, including the Pima Indians and Aboriginal Canadians in Saskatchewan, where maternal GDM is highly prevalent.[5,6] The lack of an association between birth weight and type 2 diabetes in our cohort may again reflect the fact that the average age was 32.1 years and around 30% of our cohort was under the age of 20.

Mean birth weights in our cohort were different from those reported for the province of Alberta as a whole, likely reflecting regional, socioeconomic and demographic variability. According to Alberta Perinatal Health Program (APHP) data in 2007, 6.6% of live births were LBW and 11.0% weighed >4 kg province-wide.[23] The rate of LBW is fairly constant across Canada, but HBW among Aboriginal Canadians occurs more frequently, with rates up to 21%.[24] This, in part, is thought to be due to a higher prevalence of GDM among Aboriginal populations.[6] In our cohort, 12.9% were LBW and 17.0% weighed >4 kg at birth; therefore, both LBW and HBW were more frequent in this rural population. This contradicts APHP data, which suggest that rates of LBW are lower in rural compared with urban areas.[23] The observed rate of recalled prematurity (12.3%) is higher in our cohort than the documented 2007 rate in Alberta (8.4%),[23] and may be contributing to LBW. Similarly, HBW may be elevated in this cohort in part because of increased rates of reported GDM (6.6%) compared with the 2006 rate in Alberta (3.9%).[23] On the basis of results of previous research,[25] we expected to see higher rates of LBW and HBW among Aboriginal subjects, but the distribution was similar among the three ethnic groups.

Our results add to a growing body of evidence pointing to a crucial role of the intrauterine environment in determining health outcomes later in life. Providing the best achievable care for pregnant rural and Aboriginal women should therefore be a health-care priority. However, it has been shown recently that, despite having presumably greater access to advanced prenatal care, First Nations infants born to mothers living in southern parts of Quebec have elevated risks of adverse birth outcomes compared with infants born in rural and remote northern communities.[25] The authors speculated that cultural barriers to access and use of advanced care facilities exist for urban First Nations individuals. Building on this, we speculate that a scarcity of cultural sensitivity on the part of health-care professionals, as well as a lack of understanding of and familiarity with the biomedical system on the part of Aboriginal individuals, may be barriers to proper maternal care. Present models of health practice have yet to recognize the influence on Aboriginal health of colonialism-based historical and social contexts.[26] Qualitative research indicates that many Aboriginal individuals do not seek care from the mainstream system for a variety of reasons that are generally culturally based.[27] In support, First Nations women have been shown to be screened for GDM significantly less compared with general population women (68.5% and 83.0%, respectively).[28] Clearly, there is a need for novel strategies to improve pregnancy care and subsequent outcomes.

There are limitations to this study that must be considered. The accuracy of recalling birth weights could be questioned; however, both maternal recall of infant birth weight and self-reported birth weight among middle-aged and elderly women have been found to be reliable for use in analysis.[29,30] However, lower accuracy for HBW and LBW has been noted,[30] and thus our results may underestimate the association between birth weight and CKD risk factors. We did not have data pertaining to gestational age and therefore could not adjust our birth weights accordingly. However, even if LBW were associated with prematurity in some cases, as prematurity itself is a recognized risk factor for hypertension, proteinuria and CKD, this would not be expected to have biased our findings.[31,32] Nevertheless, there is the possibility of misclassification of birth weight group owing to the lack of gestational age data. Another potential weakness in this study is that proteinuria was defined by a single positive test using either dipstick or ACR. It is possible that subjects with microalbuminuria may have been missed with dipstick testing, but the fact that proteinuria correlated with hypertension suggests that it was likely to be true; however, the association with LBW that remained after controlling for hypertension suggested an independent effect of LBW. The impact of subjects being volunteers on outcomes is difficult to assess. It is likely that the study was attended by a higher number of individuals who were concerned about their health or their children's health, possibly enriching for LBW and HBW individuals. This may have affected the birth weight prevalences in this cohort, but is unlikely to have an impact on the relationship of birth weight with disease outcomes. More universal screening programs in greater numbers of individuals are required to address these questions. Proteinuria was not confirmed with further testing.

In conclusion, in a cohort of rural, mainly Aboriginal Canadians, we show that high or LBWs are associated with proteinuria and overweight/obesity in later life. Specifically, HBW is associated with an increased risk for overweight/obesity and LBW is associated with more proteinuria. Birth weight therefore appears to be a significant risk factor for renal disease and obesity, independent of hypertension or type 2 diabetes. Importantly, birth weight is a risk factor that can be positively affected with adequate prenatal care and

maternal education. In addition, close follow-up of infants who had been of low or HBW, as well as early dietary education and emphasis on physical activity, may prevent development of additional risk factors such as overt hypertension, diabetes and obesity and reduce the complications of renal and cardiovascular disease in later life.

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