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Noise exposure during the first trimester and the risk of gestational diabetes mellitus

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Abstract
Gestational diabetes mellitus (GDM) is a form of diabetes that affects pregnant women. GDM tends to resolve after delivery, but has an impact on the health of the mother and her offspring. Considering the potential association between noise and diabetes and the susceptibility of the pregnant state to diabetogenesis, noise pollution may be associated with the risk of GDM; however, there is no evidence of the effect of noise pollution on GDM. In this study, we investigated the association between residential exposure to noise during the first trimester and incidence of GDM using the National Health Insurance Service-National Sample Cohort (NHIS-NSC), a representative sample of South Koreans. We analyzed the National Health Insurance Service–National Sample Cohort (2002–2013), a population-wide health insurance claim data. Study population was a total of 18 165 pregnant women. GDM was defined as ICD-10 code O244, and noise exposure levels were categorized as daytime (07:00–19:00) and nighttime (23:00–07:00). Other known risk factors for GDM were age, income, residential area, physical activity, smoking, drinking, blood sugar levels, and body mass index before getting pregnant. The study population included 18 165 pregnant women, of which 8.8% developed gestational diabetes. After adjustment, the adjusted OR (95% CI) for GDM associated with 1 dB increase in nighttime noise was 1.07 (95% CI: 1.05–1.10). Compared with the reference group (Quartile 1), the adjusted ORs for GDM in those exposed to the highest quartile of noise exposure (Quartile 4) was 1.61 (95% CI: 1.38–1.87) at nighttime noise. However, no significant association was observed between daytime noise exposure (07:00–19:00) and the incidence of GDM. We observed that the odds of gestational diabetes during the first trimester was 1.6 times higher for pregnant women exposed to elevated nighttime noise compared to similar women exposed to normal baseline noise levels in South Korea. Although this finding should be replicated, residential noise exposure at night may be a diabetogenic risk for pregnant women.

Introduction
Noise, defined as unwanted sound, is an emerging environmental health hazard. A recent report by the World Health Organization estimated traffic noise to be the second biggest environmental stressor globally, next only to the air pollution. Further, it accounts for at least one million healthy life-years lost every year owing to ill-health, disability, or premature death in western European countries alone [1]. There is increasing evidence connecting noise pollution to specific health risks, ranging from mere annoyance to hearing loss, sleep disturbance, cognitive impairment, and cardiovascular disease [2–4]. Recent evidence implicates noise exposure as a risk factor for type 2 diabetes mellitus (T2DM) [5–7]. A cross-sectional study by Rhee et al (2008) demonstrated that populations living near a fighter jet airbase had a higher prevalence of T2DM (p = 0.044) compared with those not exposed to any aircraft noise [5]. Cohort studies showed a significant association of the incidence of T2DM with residential noise exposure at
the time of diagnosis or during the five years preceding
the diagnosis [6] or with residential traffic intensity
[7]. A recent meta-analysis estimated that people
exposed to high residential noise levels (Lden > 60 dB)
had a 19%–22% higher risk of T2DM than did those
exposed to lower noise levels (Lden < 60–64 dB) [8].
The mechanism underlying this possible relationship
remains unclear but can be putatively explained by
linking noise-induced stress responses to the increased
risk of cardiovascular diseases [6, 8–10].

Gestational diabetes mellitus (GDM) is a form of
diabetes that affects pregnant women [11]. GDM
tends to resolve after delivery, but has an impact on the
health of the mother and her offspring. Affected
women are seven times more likely to exhibit
abnormal glucose tolerance or T2DM later in life
[12]. A review of existing studies suggested that
offspring of women with GDM have an increased risk
of all cardiorenal metabolic syndrome components
[13]. In the USA, children born to mothers with GDM
had a higher risk of overweight/obesity at age 7,
compared with offspring of mothers without GDM
[14]. In a study of Pima Indians, offspring (aged 20–24
years) of mothers with impaired glucose tolerance
during pregnancy had a substantially increased
cumulative risk of T2DM [15].

Several traditional risk factors, such as a family
history of diabetes, increased maternal age, obesity,
nonwhite ethnicity, lack of physical activity, and
cigarette smoking, are associated with an increased risk
of GDM [15, 16]. Some putative environmental risk
factors (such as exposure to metal, phthalate,
endotoxin, and air pollutants) also increase the risk
of GDM or impaired glucose tolerance [17–19].

Considering the potential association between noise
diabetes [6, 8] and the susceptibility of the
pregnant state to diabetogenesis, noise pollution may
be associated with the risk of GDM; however, there is
no evidence of the effect of noise pollution on GDM.

The objective of this study was to investigate
whether residential exposure to noise is associated
with GDM incidence. As GDM occurs typically during
the second or third trimester [11], it is especially
critical to evaluate noise exposure during the first
trimester of pregnancy when targeting at-risk GDM
pregnant women. Specifically, we assessed the potential
association between exposures to daytime and
nighttime noise during the first trimester and first
GDM events using the National Health Insurance
Service–National Sample Cohort (NHIS–NSC).

Methods

Data source and study population
NHIS of Korea has a compulsory social insurance
program that covers the entire population, with
assistance from government subsidies. NHIS is a
comprehensive source of medical claims data,
including personal information, medical examination
and treatment, prescription drugs, medical costs, and
diagnostic codes, as per the International Classification
of Diseases (ICD).

NHIS–NSC (project number: NHIS-2016-2-0081) is a population-based cohort extracted from the NHIS
database. To ensure the representativeness of the
Korean population, a stratified random sampling
design was used based on age, sex, income, residential
area, and annual medical expenses. A total of 1025,340
subjects were randomly sampled from cohort (ap-
proximately 2.2% of the total eligible population) in
2002 and the subjects followed up until 2013 [20].

From this sample, we initially included 245,405
women who were age capable (20–49 years) of
pregnancy in 2006, which was the first year of
follow-up. Among them, 43,251 women with medical
conditions of delivery (ICD-10 and O80–84) during
the follow-up period (2006–2013) were included in
this study. We further excluded 25,086 women who did
not have health examination-related data (n = 21,620)
and health behavior-related data (n = 1277) and
women who had diabetes (ICD-10, E10, E11, E12, and
E14) at the pre-pregnancy stage (n = 2189). Finally,
the remaining 18,165 women were assessed as the
study population.

Variables
GDM was defined as glucose intolerance that was not
present or recognized prior to pregnancy [21].
Diagnosis of GDM was based on the presence of
two or more of the following factors, according to the
National Diabetes Data Group criteria [22]. Its
considering ICD-10 code is O244.

Fasting serum glucose concentration ≥105 mg
\(\text{dL}^{-1}\)

1 h serum glucose concentration ≥190 mg \(\text{dL}^{-1}\)
2 h serum glucose concentration ≥165 mg \(\text{dL}^{-1}\)
3 h serum glucose concentration ≥145 mg \(\text{dL}^{-1}\)

Although many risk factors affect the development
of GDM [15, 16], data for all variables were not
available. The following baseline characteristics were
included as risk factors for GDM and were controlled
in the statistical model. Age was divided into five year
groups (20–24 years, 25–29 years, 30–34 years, 35–39
years, 40–44 years, and 45–49 years). Income was
based on household income relative to the median
(<25%, 25%–50%, 50%–75%, and >75%). Our data
consisted of a total of 254 administrative districts,
which included provincial-level divisions (i.e. special
city and metropolitan cities) and municipality-level
divisions (i.e. cities, counties, and districts). The
residential area was classified as an ‘urban area’ (special
city and metropolitan cities) or a ‘rural area’ (cities,
counties, and districts). Health behaviors included
smoking history (current, former, or never smoker)
and the status of current alcohol drinking (yes or no).
Amount of exercise was determined with the question
‘how many times a week do you exercise enough to
work up a sweat? The responses included none, 1–2 times, 3–4 times, 5–6 times, and almost every day. We categorized none as ‘no’ and 1–2 times or more as ‘yes.’ Blood glucose levels at pre-pregnancy were categorized as $\leq 125$ mg dL$^{-1}$ or $>125$ mg dL$^{-1}$. Body mass index (BMI) was calculated according to weight and height measurements, and patients were categorized as follows: underweight (BMI $<18.5$ kg m$^{-2}$), normal weight (BMI $18.5–24.9$ kg m$^{-2}$), overweight (BMI $25.0–29.9$ kg m$^{-2}$), or obese (BMI $\geq 30.0$ kg m$^{-2}$).

Estimation of individual exposure to noise: daytime and nighttime

Data pertaining to noise exposure were obtained from the National Noise Information System (NNIS) (www.noiseinfo.or.kr/eng/index.jsp). Regarding the specific requirements for noise data collection accuracy and uniformity, measurements were based on the Act on Environmental Testing and Inspection and observed the Official Test Standard for Noise and Vibration, which specifies the requirements for noise measurement accuracy and uniformity [23]. Briefly, noise was measured in areas such as cities, where it is important to maintain normal living conditions with considering the population and city size. In each measurement area, noise was measured on weekdays, and the measured noise levels were calculated as the arithmetic mean values at each point. Daytime noise was measured at least four times at intervals of $\geq 2$ h, and nighttime noise was measured at least two times at intervals of 2 h. The noise measurement equipment was designed as exhibiting a Class 2 noise level or equivalent performance (Korea standard C 61672-1 International Electrotechnical Commission). Measurements made with this scale were expressed in units of dB (A), and the measurement range was 35–130 dB [23].

The NNIS provides national environmental noise level data measured through automatic or manual processes. However, as automatic noise measurement is performed in relatively few areas, we used noise data that were manually obtained at 1286–1372 monitoring sites during the study period. Data for the period from January 2006 to December 2013 were downloaded and used to calculate the monthly mean noise levels in the daytime (07:00–19:00) and nighttime (23:00–07:00).

Noise levels in unmonitored regions were estimated using geographic information systems (GIS) tools. The Kriging geo-statistical method was used for spatial data interpolation. We applied Empirical Bayesian Kriging (EBK), which automatically calculates the Kriging parameters using a process of subsetting and simulations. EBK is a reliable automatic interpolator that allows relatively more accurate predictions of non-stationary data from small datasets compared with other Kriging methods [24]. Furthermore, to calculate the monthly daytime and nighttime noise levels in each region, zonal statistics from three layers were used: zonal, value, and output layers. A zone was defined as a region of areas with identical values and comprised 254 administrative districts. The value layer contained the input value used to calculate the output for each zone and comprised 66304 cells with noise exposure values. The statistic applied to the value input led to the calculation of the mean noise exposure per administrative district as the output layer. The extracted regional noise data were matched with the individual administrative district codes (based on home addresses) to assign individual noise exposure levels during the first trimester.

Statistical analysis

Chi-square tests were used to assess between group differences with respect to variables of interest in pregnant women with and without GDM. Noise levels were treated as both a continuous and a categorical variable. We divided the study population by quartiles of daytime (Quartile 1, $<60.27$ dB; Quartile 2, 60.27–61.5 dB; Quartile 3, 61.5–62.79 dB; and Quartile 4, $\geq 62.79$ dB) and nighttime (Quartile 1, $<54.22$ dB; Quartile 2, 54.22–55.84 dB; Quartile 3, 55.84–57.34 dB; and Quartile 4, $\geq 57.34$ dB), respectively.

We performed unadjusted and multivariate-adjusted logistic regression analyses to assess the likelihood of GDM and stratified the findings by an increase in 1 dB or by the quartiles of noise levels during the first trimester. Odds ratio (OR) with corresponding 95% confidence intervals (CIs) for the likelihood of GDM with an increase in 1 dB of daytime and nighttime noise, respectively, were generated. The ORs for GDM in each quartile (Quartile 2–4) were calculated using the Quartile 1 as the reference group. We also considered a generalized additive model (GAM), which visualizes an unconstrained picture of the association between noise exposure and GDM by incorporating nonlinear effects. This type of model consequently helps uncover hidden patterns in the observed data and thus reduces the risk of over-fitting [25]. In these two regression models, the independent variable was the daytime and nighttime noise exposure levels, and the dependent variable was the GDM prevalence. Potential confounding variables included age, income, residential area, smoking, exercise, alcohol drinking, blood glucose levels, and BMI categories. These sentences have been included in the revised manuscript. All analyses were performed using SAS 9.2 software (SAS Institute, Cary, NC, USA), and the statistical significance level was set at $\alpha = 0.05$.

Results

Table 1 shows study population by presence of GDM. Of the 18165 participants, 1596 women (8.8%) had developed GDM. Pregnant women who had GDM were more likely to be older, have low incomes, live in
urban areas, and to have high blood glucose levels (>125 mg dL⁻¹). There was no significant difference with respect to health behaviors (i.e. exercise, smoking, and alcohol drinking) and BMI between the two groups.

Figure 1 displays the mean noise levels based on the presence of GDM and the prevalence of GDM by quartiles of noise exposure in the first trimester. During the study period, the mean daytime and nighttime noise levels (min–max) were 61.58 dB (55.49–67.33 dB) and 55.66 dB (43.77–62.16 dB), respectively. The correlation coefficients of the daytime and nighttime noise levels were highly significant (r = 0.8356; p < 0.0001). Participants with GDM were exposed to significantly higher mean daytime (61.67 vs. 61.57 dB; p = 0.0454) and nighttime noise levels (56.11 vs. 55.62 dB; p < 0.0001) during the first trimester compared with those without GDM. The proportion of GDM women was the largest in the high noise quartiles (Quartile 4) in both daytime and nighttime. The prevalence of GDM was significantly increased with increase in the quartiles of the first trimester nighttime noise (p < 0.0001), but not daytime (p = 0.2573).

Table 2 shows the ORs for GDM by noise exposure in the first trimester. The following percentages of pregnant women were classified by daytime noise quartiles: 24.19% in Quartile 1 (the lowest exposure), 23.93% in Quartile 2, 24.69% in Quartile 3, and 27.19% in Quartile 4 (the highest exposure). There appeared to be no linear increase in GDM patients with increasing daytime noise levels. In contrast, the percentage of pregnant women with GDM gradually increased with increasing nighttime noise, with 20.30%, 22.56%, 22.68%, and 32.46% classified as Quartile 1, 2, 3, and 4, respectively. After adjustment for all potential confounding variables, ORs for GDM were significantly increased with increase in 1 dB of nighttime noise (OR = 1.07; 95% CI: 1.05–1.10) in the first trimester; daytime noise did not associated with the incidence for GDM. Compared with the reference group (Quartile 1), the adjusted ORs for GDM in those exposed to the highest quartile of noise exposure (Quartile 4) was
Figure 1. Mean noise levels based on the presence of GDM and the prevalence of GDM by quartiles of noise exposure in the first trimester in South Korea (2006–2013).

Table 2. Odds ratio (OR) of gestational diabetes mellitus in relation of quartiles of noise levels in the first trimester in South Korea (2006–2013).

<table>
<thead>
<tr>
<th>Noise levels</th>
<th>no. of with GDM / without GDM</th>
<th>Crude OR (95% CI)</th>
<th>p-trend</th>
<th>Adjusted OR (95% CI)</th>
<th>p-trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime noise (dB)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 dB increase</td>
<td>1.03 (1.01–1.06)</td>
<td>0.0346</td>
<td>1.01 (0.98–1.04)</td>
<td>0.6794</td>
<td></td>
</tr>
<tr>
<td>Quartiles</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (&lt;60.27)</td>
<td>386/4168</td>
<td>Reference</td>
<td>0.2576</td>
<td>Reference</td>
<td>0.8113</td>
</tr>
<tr>
<td>Q2 (60.27–61.50)</td>
<td>382/4152</td>
<td>0.99 (0.86–1.15)</td>
<td>0.96 (0.83–1.11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3 (61.51–62.78)</td>
<td>394/4105</td>
<td>1.04 (0.9–1.20)</td>
<td>0.97 (0.83–1.12)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4 (≥62.79)</td>
<td>434/4144</td>
<td>1.13 (0.98–1.31)</td>
<td>1.02 (0.88–1.18)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nighttime noise (dB)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 dB increase</td>
<td>1.10 (1.08–1.13)</td>
<td>&lt;.0001</td>
<td>1.07 (1.05–1.10)</td>
<td>&lt;.0001</td>
<td></td>
</tr>
<tr>
<td>Quartiles</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (&lt;54.22)</td>
<td>324/4247</td>
<td>Reference</td>
<td>&lt;.0001</td>
<td>Reference</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Q2 (54.22–55.84)</td>
<td>360/4235</td>
<td>1.11 (0.95–1.30)</td>
<td>1.10 (0.94–1.28)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3 (55.85–57.33)</td>
<td>362/4204</td>
<td>1.13 (0.97–1.32)</td>
<td>1.05 (0.89–1.23)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4 (≥57.34)</td>
<td>550/3883</td>
<td>1.86 (1.61–2.14)</td>
<td>1.61 (1.38–1.87)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Adjusted for age (20–24 years, 25–29 years, 30–34 years, 35–39 years, 40–44 years, or 45–49 years), household income relative to the median (<25%, 25%–50%, 50%–75%, or >75%), residence area (urban or rural), smoking history (current, former, or never smoker), exercise (yes or no), alcohol drinking (yes or no), blood glucose levels at pre-pregnancy (≤125 mg dL⁻¹ or >125 mg dL⁻¹), and BMI (underweight, <18.5 kg m⁻²; normal weight, 18.5–24.9 kg m⁻²; overweight, 25–29.9 kg m⁻²; or obese, ≥30.0 kg m⁻²).
1.61 (95% CI: 1.38–1.87) at nighttime noise, but not daytime (OR = 1.02, 95% CI: 0.88–1.18).

Figure 2 displays the adjusted association (spline function) plots of the GMD risk with daytime and nighttime noise. Overall, the results show a linear association between these variables; in particular, the odds of GDM were noticeably higher with >55 dB at nighttime.

Discussion

In this study, we investigated the association between residential noise exposure and the incidence of GDM. We found that first trimester exposure to nighttime noise (23:00–07:00) was significantly associated with an increased risk for GDM. Specifically, the adjusted OR (95% CI) for GDM associated with 1 dB increase in nighttime noise was 1.07 (95% CI: 1.05–1.10). The adjusted OR for GDM in the highest quartile (Quartile 4) of nighttime noise (using Quartile 1 as reference was 1.61 (95% CI: 1.38–1.87). Of note, no significant association was observed between daytime noise exposure (07:00–19:00) and the incidence of GDM.

To date, no studies have examined the relationship between noise exposure and GDM. However, previous studies have suggested an association of exposure to noise with the risk of T2DM [4, 6]. The results of some studies implicated noise exposure from airports and roadways in the development of T2DM [6–8], although some inconsistent data were reported [26]. The mechanism by which exposures to noise affect the progression of T2DM remains unclear. However, plausible biological explanations have been suggested on the possibility that noise-induced stress responses can activate the hypothalamic–pituitary–adrenal axis and the sympathetic autonomic nervous system, which in turn may result in dysregulation of β-cell function, insulin sensitivity, and glucose tolerance [3, 8]. Studies have demonstrated that various forms of noise exposures from experimental settings to road traffic or aircraft cause significant increase in corticosterone, cortisol, and catecholamine levels in both humans and animals [3, 27–29]. Further studies are needed to confirm the contribution of noise-induced stress to subsequent development of T2DM. Moreover, given the adverse or mildly harmful health effects (i.e. gestational hypertension, small-for-gestational-age newborn, and congenital malformations) of noise on pregnancy [30], additional studies are required to further confirm whether environmental noise exposure increases the risk of diabetes in pregnant women, as was consistently observed in our study.

Of note, our results suggested that residential exposure to night noise in the first trimester was a strong correlate of GDM, while no such association was observed for daytime noise exposure. This is in line with the suggestions that health risks may differ based on the time of exposure [31] and nighttime noise may be particularly relevant to certain health issues [32, 33]. For example, the association between environmental noise and hypertension was stronger for nighttime exposure [34–36].

The specific mechanism of nighttime noise-induced health problems is not clear. Interference of sleep is thought substantially contribute to the adverse effects of chronic exposure to nighttime noise [3, 37]. Admittedly, our data did not provide information on sleep quality. However, considering that approximately 63% of our study population was exposed to nighttime noise of > 55 dB (categorized as being of public health significance in the European guidelines for nighttime noise) [37], our study participants were speculated to have a high possibility of poor sleep quality. In general, pregnant women undergo profound endocrine and metabolic changes (i.e. impaired insulin sensitivity, increased β-cell function, and moderate elevation of blood glucose level) to accommodate the needs of the maternal-fetal unit [38]. Nevertheless, pregnant women who develop GDM are more likely to have a greater severity of insulin resistance [38].
together, exposures to chronic and high levels of nighttime noise could stimulate secretion of stress hormones (i.e. cortisol and catecholamines) and could induce sleep disturbance, which can further impair endocrine and metabolic parameters including insulin and glucose metabolism [39–42]. Thus, nighttime noise exposure may be a high-risk condition for the subsequent development of GDM.

To the best of our knowledge, this is the first study to investigate the association between nighttime noise exposure during the first trimester and the risk for GDM. However, several critical limitations should be considered. Most important limitation is the noise exposure assessment. Noise exposure levels did not monitored directly by personal samplers, and we obtained the exposure data from a national agency of Korea. We performed the Kriging interpolation technique to estimate noise levels at unmonitored locations. Currently, no standard procedure exists for the spatial interpolation of noise data. Although the Kriging is among the most widely used interpolation methods in air pollution studies [43], it has been described as a superior method for predicting noise properties [44]; therefore, it may be appropriate for the interpolation of our noise data. Furthermore, since individual noise exposure was estimated using the GIS based on administrative district codes of their home address, these do not necessarily reflect the exact individual exposure to noise. Secondly, we analyzed a representative population-based cohort data of the NHIS-NSC [20]. Although this cohort included a large-scale sample (i.e. >1 million participants) selected using a randomized sampling design, it is not a specific-purpose cohort, and information about a specific disease may be insufficient [20]. For example, a few cells of women with GDM had very low numbers (e.g. age group 45–49 years (n = 1), blood sugar >125 (n = 15), and obesity (n = 17)), as shown in table 1. In addition, the disease codes were entered into health insurance claims associated with patient services, and thus, the codes used in this cohort may not accurately reflect the actual patients’ disease status. This is an inherent limitation of studies based on insurance databases. It is also difficult to guarantee representation during follow-up years. In addition, we adjusted variables for socioeconomic characteristics, health behavior, obesity, and blood glucose at baseline. However, we did not adjust for other potential risk factors, including genetics, family history of diabetes, and environmental exposures (i.e. chemicals and air pollution) owing to a lack of data availability on these variables.

Environmental noise summarizes noise pollution from external sources including transport, industrial and recreational activities [45]. To minimize the health effects of environmental noise exposure, efforts to reduce the frequency and strength of environmental noise should be accompanied; this includes legal, institutional, and engineering improvements. Specifically, environmental noise regulations can be established for residential areas, improvements can be made to housing and construction guidelines to eliminate noise transmission, low noise vehicles can be introduced, or road traffic can be improved. Personal efforts (e.g. the use of interior sound insulation and sound absorption materials or of noise protectors such as earplugs and earmuffs) would be simultaneously performed. Moreover, regarding higher-than-appropriate workplace noise levels, more attention may be needed regarding the working environments of pregnant women.

Conclusion

In conclusion, we observed a significant association of nighttime noise exposure during the first trimester with the incidence of GDM. Our findings add to the available body of evidence linking noise exposure with increased risk of diabetes, and suggest that residential noise exposure at night might be a diabetogenic risk for pregnant women. Regulations and actions to reduce and manage noise from various sources are critical for the prevention of this health risk. These efforts need more attention with the aim of improving the protection of this vulnerable population (i.e. pregnant women) from noise exposure.

Acknowledgments

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