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Original article

Egg consumption and risk of type 2 diabetes among African Americans: The Jackson Heart Study

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SUMMARY

Background & aims: Type 2 diabetes (DM) disproportionally affects African Americans. Data on the association between egg consumption and risk of DM are sparse. We sought to examine whether egg consumption is associated with the prevalence and incidence of DM among African Americans.
Methods: We analyzed baseline data from 4568 participants of the Jackson Heart Study. Egg consumption was obtained using a food frequency questionnaire designed for this population. We used generalized estimating equations to calculate adjusted prevalence ratios of DM and Cox regression to estimate hazard ratios of DM with corresponding 95% confidence intervals (CI).
Results: The average age was 55 ± 13 years and 64% of subjects were women. The median frequency of egg consumption was 2/week for men and 1/week for women. The prevalence of DM was 22% overall (21% of men and 23% of women). Multivariable adjusted prevalence ratio [PR (95% CI)] for DM were: 1.00 (ref), 1.14 (0.90–1.44), 1.33 (1.04–1.70), 1.33 (1.06–1.68), 1.26 (0.99–1.61), and 1.52 (1.17–1.97) for egg consumption of <1/month, 1–3/month, 1/week, 2/week, 3–4/week, and 5+/week, respectively, p for linear trend 0.0024. Corresponding multivariable adjusted hazard ratios were 1.00 (ref), 0.88 (0.65–1.19), 0.94 (0.68–1.30), 0.91 (0.66–1.25), 1.11 (0.81–1.52), and 1.17 (0.81–1.70), respectively, during a mean follow up of 7.3 years (p for linear trend 0.22).
Conclusions: While egg consumption was positively associated with prevalent DM, prospective analysis did not show an association of egg intake with incidence of DM among African Americans.
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1. Introduction

As a major risk factor for cardiovascular disease (CVD), type 2 diabetes (DM) is a major health issue worldwide [1–3]. It is anticipated that 440 million individuals will be diagnosed with DM by 2030 [2]. Global healthcare expenditures due to DM are projected around 490 billion dollars by 2030 [3]. The burden of DM disproportionally affects African-Americans. In 2009, the risk of death per 100,000 due to DM was 44.2 for black men (vs. 23.3 for white men) and 35.9 for black women (vs. 15.7 for white women) [4].

Previous research has identified modifiable lifestyle factors such as diet and physical activity as important determinants of DM [5]. However, the association between different macronutrients and risk of DM is not consistent. For example, among saturated fatty acids, while short-chain saturated fatty acids have been associated with a higher risk of DM, odds chain and long-chain saturated fatty acids have been related with a lower risk of DM [6–8]. In addition, the EPIC cohort reported that animal but not vegetable proteins

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were associated with a higher risk of DM [9] whereas a meta-analysis of three studies showed no association of animal proteins with DM [10].

Among individual foods, eggs could play a unique role among dietary factors affecting the risk of DM, partly because they are rich in proteins, cholesterol, and other nutrients. Current data on the association of eggs with DM have been inconsistent, ranging from no association in Japanese [11] and non-Hispanic white adults [12,13] to a higher risk of DM with frequent intake of eggs in Chinese [14] and other populations [15–18]. In two previous meta-analyses, the highest category of egg consumption was associated with a 42%–68% higher risk of DM [19,20]; however most of those data were derived from non-Hispanic whites, and it is unclear whether egg consumption is associated with a higher risk of DM in African Americans. Thus, the current paper sought to examine the association of egg consumption with prevalent and incident DM among African American participants of the Jackson Heart Study (JHS).

2. Methods

2.1. Study population

The present analysis used JHS data collected during the baseline examination (2000–2004). Detailed description of the JHS has been previously published [21–25]. Of the 5301 JHS participants who completed a baseline clinic examination, we excluded 248 who had missing data on egg consumption; one person with type 1 DM; 16 with restricted informed consent; 422 with energy intake outside of normal ranges (600–4000 kilocalories per day); and 46 with missing information on DM status. Thus, current analyses are based on 4568 individuals with complete data for cross-sectional analyses and 3564 subjects for prospective analyses after exclusion of prevalent DM (n = 1004). Each participant gave written informed consent and the study protocol was approved by the institutional review boards of all institutions involved.

2.2. Assessment of egg consumption

Egg consumption was assessed using a 158-item food frequency questionnaire (FFQ) [26] that has been validated in this cohort [27]. Participants reported their frequency of egg consumption, including fried and scrambled eggs, as never; less than once per month; 1/month; 2–3/month; 1/week; 2/week; 3–4/week; 5–6/week; 1/day; or 2/day. Adjacent categories were combined into <1/month; 1–3/month; 1/week; 2/week; 3–4/week; and 5+/week to allow for stable estimate in current analyses.

2.3. Ascertainment of DM

Prevalent DM was defined as fasting glucose ≥126 mg/dL, hemoglobin A1C ≥ 6.5%, or current use of insulin or oral hypoglycemic agents at baseline. Similar criteria were used to define incident DM during follow up among participants that were free of DM at baseline. Since we did not have information on the exact date of diagnosis of DM, we assigned the JHS visit date following the initial diagnosis of DM as date of DM occurrence.

2.4. Other variables

Assays and procedures for measuring fasting whole blood, plasma, and serum-derived measurements in the JHS have been previously described [21]. At JHS visit 1, we collected information on demographic and anthropometric variables, cigarette smoking, physical activity, education, alcohol consumption, history of hypertension, CVD, LDL- and HDL-cholesterol, triglycerides, and diet. Intakes of fruit and vegetables, red meat, fish, trans fat, total energy, dietary fiber, and dietary magnesium were used as covariates. Physical activity was measured as a sum of four index scores (active living, work/occupational, home life, and sports) [28].

2.5. Statistical analysis

Baseline characteristics within each category of egg consumption are presented as mean ± standard deviation (SD) for continuous variables with Gaussian distribution (otherwise median with inter-quartile range), and proportions for categorical variables. We used generalized estimating equations to calculate prevalence ratio (PR) for cross-sectional analyses and Cox proportional hazard model for prospective analyses along with corresponding 95% confidence limits. Person-time of follow-up was computed from baseline until the first occurrence of incident diabetes, death, or last contact. We built sequential models based on a priori knowledge. In model 1, we adjusted for age (≤45, <45–55, <55–65, <65) and sex. Model 2 was additionally adjusted for BMI (<25, 25–<30, 30–<35, 35+), smoking status (never, former, current), alcohol intake in the past 12 months (yes/no), physical activity score (tertiles), and education (less than high school/high school or some college/associates degree or higher). The final model adjusted for variables in model 2 plus energy intake (tertiles), red meat (tertiles), dietary fiber (tertiles), dietary magnesium (tertiles), fruit and vegetable intake (tertiles), dietary trans fat (tertiles), waist circumference (tertiles), history of hypertension (yes/no), and history of CVD (yes/no). We obtained a p-value for linear trend by including an ordinal variable that was assigned to frequency of egg consumption in the regression model. Cox proportional hazard model assumption were tested using product term of egg frequency and log-transformed person-time and were met (all $p > 0.05$).

In secondary analyses, we stratified the main analysis by sex, BMI, and age and also examined whether egg consumption was associated with baseline measures of insulin resistance and A1C by computing least squares means for logarithmic-transformed A1C, homeostatic model assessment-insulin resistance (HOMA-IR), and homeostatic model assessment-beta cell function (HOMA-B). Log-transformation was necessary due to non-Gaussian distribution of HOMA and A1C measures. All analyses were completed using Statistical Analysis Systems, version 9.3 (SAS Institute, Cary, North Carolina) with alpha level of 0.05.

3. Results

Baseline characteristics of the 4568 African Americans (Table 1) show a mean age of 55 ± 13 years (range 21–95), with 64% women. The prevalence of DM was 22% (21% of men and 23% of women). Compared to egg consumption of < once per month, higher egg consumption was associated with male sex, current smoking, lower educational attainment, alcohol consumption, higher waist circumference, lower HDL, and consumption of red meat, fish, bacon, saturated and trans fats (Table 1). Egg consumption was correlated with saturated fat (Spearman r = 0.37), energy intake (r = 0.32), trans fatty acids (r = 0.25), processed meats (r = 0.21), dietary magnesium (r = 0.18), and fiber (r = 0.13).

3.1. Cross-sectional analyses (eggs with prevalent DM and biomarkers)

In a model adjusting for age and sex, prevalence ratios (95% CI) for DM were 1.00 (ref), 1.06 (0.85–1.32), 1.28 (1.02–1.60), 1.29 (1.05–1.60), 1.25 (1.00–1.56), and 1.54 (1.22–1.94) from the lowest to highest categories of egg consumption (p for linear trend 0.0001, Table 2). These findings persisted and were not substantially altered

Table 1
Characteristics of the 4568 Jackson Heart Study participants by frequency of egg consumption[a].

	Egg Consumption Categories						p for linear trend
	<1/month (n = 742)	1–3/month (n = 958)	1/week (n = 698)	2/week (n = 881)	3–4/week (n = 781)	5+/week (n = 508)	
Age (y)	57 ± 12	54 ± 13	55 ± 12	55 ± 13	55 ± 12	57 ± 13	0.43
BMI (kg/m^2)	31 ± 7	31 ± 7	32 ± 8	32 ± 7	32 ± 7	32 ± 7	0.01
Male (%)	23	32	37	37	41	50	<0.0001
Smoking (%)							
Never	71	69	71	73	65	61	0.0013
Past	20	20	18	16	21	24	0.23
Current	7.6	12	11	11	14	15	0.002
Education (%)							
< High School	19	16	16	17	17	26	0.02
High School/GED/Some College	42	37	41	42	44	45	0.02
College/Assoc. deg or higher	39	47	43	42	39	29	<0.0001
Drank alcohol in past year (%)	33	49	46	45	48	46	0.0001
Hypertension (%)	67	58	61	63	66	69	0.02
CVD (%)	11.2	7.8	9.6	10	12	13	0.02
LDL (mg/dL)	128 ± 38	128 ± 37	127 ± 37	128 ± 36	125 ± 35	127 ± 35	0.19
HDL (mg/dL)	53 ± 15	53 ± 15	51 ± 14	52 ± 14	51 ± 15	51 ± 14	0.0002
Triglycerides (mg/dL)	107 ± 63	103 ± 69	104 ± 62	109 ± 71	103 ± 78	111 ± 80	0.25
Waist Circumference (cm)	99 ± 15	99 ± 16	100 ± 17	101 ± 17	102 ± 16	103 ± 15	<0.0001
Physical Activity Score	8.0 ± 2.5	8.5 ± 2.5	8.6 ± 2.5	8.3 ± 2.6	8.4 ± 2.6	8.0 ± 2.8	0.77
Fruit and Vegetables (servings/day)	2.8 [1.8–4.3]	2.7 [1.7–3.9]	2.8 [1.8–4.1]	3.4 [2.3–4.7]	3.2 [2.2–4.5]	3.5 [2.4–5.0]	<0.0001
Red Meat (g/day)	7.8 [3.0–20]	8.1 [3.5–16]	12.4 [5.0–24]	14.5 [7.2–27]	15.3 [7.7–29]	21 [10–38]	<0.0001
Fish (g/day)	8.6 [3.0–20.7]	9.7 [4.3–21.9]	13.8 [5.3–27.4]	12.0 [5.6–27.1]	13.8 [5.3–27.1]	10.8 [4.3–24.7]	<0.0001
Saturated Fat (g/day)	18 [12–26]	19 [14–27]	23 [16–31]	26 [18–35]	29 [21–38]	34 [26–44]	<0.0001
Trans Fat (g/day)	3.2 [2.1–4.8]	3.4 [2.3–4.8]	3.7 [2.7–5.7]	4.4 [2.9–6.2]	4.6 [3.2–6.6]	5.2 [3.5–7.3]	<0.0001
Dietary Cholesterol (mg/day)	162 [109–243]	190 [145–255]	257 [192–327]	334 [260–433]	439 [345–548]	657 [478–804]	<0.0001
Dietary Magnesium (mg/day)	251 [191–326]	244 [191–319]	270 [215–354]	286 [225–359]	298 [229–371]	315 [238–388]	<0.0001
Dietary fiber (g/day)	13 ± 6	12 ± 6	14 ± 6	14 ± 6	14 ± 6	15 ± 6	<0.0001
Calories (kcal/d)	1644 ± 709	1655 ± 703	1869 ± 746	2041 ± 773	2154 ± 780	2348 ± 786	<0.0001

[a] Mean ± SD for continuous variables with Gaussian distribution or median [interquartile range] if not normally distributed.

after additional adjustment for smoking status, alcohol consumption, BMI, physical activity score, education, energy intake, intake of red meat and fruit and vegetables, trans fat, dietary magnesium, and fiber, and history of hypertension and CVD (Table 2).

In secondary analysis, we observed similar egg-DM associations in men (p-trend 0.05) and women (p-trend 0.02), Table 3. Neither sex (p-interaction 0.53), nor age (p-interaction 0.88) or BMI (p-interaction 0.20) modified the association of egg consumption with prevalent DM. Lastly, egg consumption was not associated with mean A1C, HOMA-IR, or HOMA-B in multivariable adjusted models with a subsample of 3178 participants without DM (Table 4).

3.2. Prospective analyses (eggs and incidence of DM)

During a mean follow up of 7.3 years, 531 new cases of DM occurred among 3564 subjects free of DM at baseline. In both crude

Table 3
Prevalence ratios (95% confidence intervals) for prevalent type 2 diabetes by egg consumption stratified by gender[a].

Multivariable adjusted Model		
Egg consumption	Male	Female
<1/month	1.00 (ref)	1.00 (ref)
1–3/month	1.78 (1.05–3.00)	0.99 (0.75–1.29)
1/week	1.68 (0.98–2.87)	1.26 (0.95–1.67)
2/week	1.71 (1.01–2.88)	1.26 (0.97–1.64)
3–4/week	1.65 (0.97–2.78)	1.19 (0.90–1.58)
5+/week	2.10 (1.24–3.57)	1.38 (1.00–1.90)
p for trend	0.048	0.019

[a] Adjusted for age, smoking, alcohol, BMI, physical activity score, education, energy intake, intake of red meat and fruits and vegetables, dietary trans fat, magnesium, and fiber, and history of hypertension and CVD. p for sex*egg interaction = 0.53.

Table 2
Prevalence ratios (95% CI) of diabetes at baseline according to egg consumption in the Jackson Heart Study (n = 4568).

Prevalence ratios (95% CI)					
Egg consumption	Cases/n[a]	Crude	Model 1[b]	Model 2[c]	Model 3[d]
<1/month	145/742	1.00	1.00	1.00	1.00
1–3/month	180/958	0.96 (0.77–1.20)	1.06 (0.85–1.32)	1.11 (0.89–1.40)	1.14 (0.90–1.44)
1/week	156/698	1.14 (0.91–1.43)	1.28 (1.02–1.60)	1.30 (1.02–1.64)	1.33 (1.04–1.70)
2/week	206/881	1.20 (0.97–1.49)	1.29 (1.05–1.60)	1.31 (1.05–1.63)	1.33 (1.06–1.68)
3–4/week	172/781	1.13 (0.90–1.41)	1.25 (1.00–1.56)	1.23 (0.98–1.55)	1.26 (0.99–1.61)
5+/week	145/508	1.46 (1.16–1.84)	1.54 (1.22–1.94)	1.43 (1.11–1.82)	1.52 (1.17–1.97)
p for trend		0.0005	0.0001	0.0042	0.0024

[a] Number of prevalent diabetes/total number of subjects in each category of egg consumption.
[b] Model 1 adjusted for age and sex.
[c] Model 2 adjusted for age, sex, smoking, alcohol, BMI, physical activity score, and education.
[d] Model 3 adjusted for variables in model 2 plus additional adjustment for energy intake, red meat (including bacon), fiber, dietary magnesium, fruit/vegetables, trans fat, waist circumference, history of hypertension, and history of CVD.

Table 4
Least squares means (standard errors) for the logarithmic-transformed A1C, HOMA-IR, and HOMA-B in 3178 non-diabetic subjects[a].

Egg consumption	A1C	HOMA-IR	HOMA-B
<1/month	1.71 (0.004)	1.10 (0.02)	5.25 (0.02)
1–3/month	1.70 (0.003)	1.11 (0.02)	5.28 (0.02)
1/week	1.70 (0.004)	1.12 (0.02)	5.28 (0.02)
2/week	1.70 (0.003)	1.10 (0.02)	5.26 (0.02)
3–4/week	1.70 (0.004)	1.08 (0.02)	5.27 (0.02)
5+/week	1.70 (0.01)	1.13 (0.03)	5.27 (0.03)
P linear trend	0.25	0.77	0.72

A1C, HOMA-IR, and HOMA-B were all logarithmically transformed.
[a] Derived from generalized linear model adjusted for age, gender, smoking, BMI, alcohol, physical activity score, and education.

and multivariable adjusted analyses, egg consumption was not associated with the risk of developing DM (Table 5). Gender, age, and BMI did not modify the relation of egg consumption with incidence of DM (all p for interaction >0.58).

4. Discussion

In this large cohort, we found a positive and linear relation between frequency of egg consumption and baseline prevalence of DM in African Americans. However, no association was observed between frequency of egg consumption and incidence rate of DM in this cohort. In a secondary cross-sectional analysis, we found no evidence for an association of egg consumption with A1C concentration or with measures of insulin resistance (HOMA-IR and HOMA-B) in participants without DM at baseline.

To our knowledge, this is the first study to examine the relation of egg consumption with prevalent and incident DM in a large cohort of African Americans. Contrary to our cross-sectional results, we did not find evidence for an association between frequency of egg consumption and the incidence of DM during a mean follow up of seven years in this cohort. A lack of an association between egg consumption and incidence of DM is consistent with results from the Cardiovascular Health Study, where egg consumption was not associated with incidence of DM among older US adults who were predominantly Caucasians [13]. Similarly, neither a large Japanese cohort of 63,466 adult men and women [11] nor a Mediterranean cohort of 15,956 adult men and women [12] found an association of egg consumption with DM with comparable follow up times. Nevertheless, our prospective findings are contrary to the health professional data showing a positive relation between egg consumption and incident DM [15]. A case-control study in a Lithuanian out-patient clinic reported a three-fold increased odds of DM comparing ≥5 eggs/week to <1 egg/week [17]. Furthermore, a prospective study of about 3000 women reported positive and graded relation of egg consumption with gestational DM (P for trend = 0.008)] [16]. A cross-sectional analysis of Chinese adults showed a two-fold higher odds of DM comparing 1+ eggs/d to <2 eggs/week (OR = 2.28 (1.14–4.54)] [14], finding that is consistent with our cross-sectional results. In a meta-analysis using prospective data from the Physicians' Health Study, the Women's Health Study, the Cardiovascular Health Study (CHS), and the Adventist Health Studies, the highest category of egg consumption was associated with 42% higher risk of DM (95% CI: 9–86%) when compared to the lowest category [19]. Another meta-analysis, which included studies in China and Lithuania, also found a positive relation of egg consumption with risk of DM [pooled RR: 1.68 (95% CI: 1.41–2.00)] [20]. Of note is that above meta-analyses did not include large recent studies with negative findings (i.e., the Japan Public Health Center-based Prospective Study with more than 63,000 people).

Several factors could account for the inconsistency of these findings with our results. First, differences in overall dietary habits (i.e., adherence to Mediterranean diet known to lower DM risk [29]) between cohorts reporting positive versus no association could partially explain the inconsistency, where the effect of a single food item such as eggs, may be insignificant when compared to the effects of an overall healthy dietary pattern. Second, differences in other lifestyle factors that are inherently associated with ethnic groups or geographic locations might account for some of the disparities. In the current study, we did not assign a high weight to cross-sectional results in the presence of prospective data since we cannot ascertain temporality in cross-sectional analyses. It is possible that subjects diagnosed with DM altered their diet voluntarily or per clinicians' advice to obtain their proteins from eggs rather than red or processed meats. Such a scenario could partially explain the positive relation observed between egg consumption and prevalent DM in our cohort.

Eggs are a good source of protein and other nutrients. However, a medium egg also contains up to 200 mg of dietary cholesterol [30]. Qiu et al. [16] reported a positive association between dietary cholesterol intake and risk of gestational DM. However, our group reported no evidence of association between dietary cholesterol and incident DM in older adults. In addition, a Japanese study [11] reported an inverse relation of dietary cholesterol with DM risk [OR = 0·68 (95% CI 0·49–0·94), comparing the fourth to the first quartile of dietary cholesterol]. A human study found that 12 weeks of a high-protein diet with eggs improved fasting blood glucose (−0.5 mmol/l) [31]. Taken together, current data do not lend support to the hypothesis that dietary cholesterol is a major culprit for the development of DM. Could choline metabolites help explain positive results of egg-DM association?

Table 5
Hazard ratios (95% CI) of diabetes according to egg consumption in the Jackson Heart Study (n = 3564).

Hazard ratios (95% CI)					
Egg Consumption	Cases/n[a]	Crude	Model 1[b]	Model 2[c]	Model 3[d]
<1/month	92/597	1.00	1.00	1.00	1.00
1–3/month	101/778	0.82 (0.62–1.09)	0.85 (0.64–1.13)	0.83 (0.62–1.11)	0.88 (0.65–1.19)
1/week	79/542	0.92 (0.68–1.24)	0.95 (0.71–1.29)	0.90 (0.66–1.23)	0.94 (0.68–1.30)
2/week	101/675	0.95 (0.71–1.26)	0.97 (0.73–1.29)	0.88 (0.65–1.18)	0.91 (0.66–1.25)
3–4/week	102/609	1.08 (0.82–1.43)	1.12 (0.84–1.49)	1.07 (0.79–1.43)	1.11 (0.81–1.52)
5+/week	56/363	1.05 (0.75–1.47)	1.09 (0.78–1.53)	1.05 (0.74–1.48)	1.17 (0.81–1.70)
p for trend		0.20	0.15	0.34	0.22

[a] Number of incident diabetes/total number of subjects at risk in each category of egg consumption.
[b] Model 1 adjusted for age and sex.
[c] Model 2 adjusted for age, sex, smoking, alcohol, BMI, physical activity score, and education.
[d] Model 3 adjusted for variables in model 2 plus additional adjustment for energy intake, red meat (including bacon), fiber, dietary magnesium, fruit/vegetables, trans fat, waist circumference, history of hypertension, and history of CVD.

Eggs are an important source of choline [32], which can be metabolized by gut bacteria to generate trimelthylamine, that can further be transformed to trimethylamine-N-oxide (TMAO) in the liver [33]. Tang et al. [34] reported a positive association of egg consumption and TMAO concentration. In a randomized controlled trial of six volunteers, higher egg yolk intake led to a graded increase in plasma and urine TMAO [32]. TMAO may increase LDL oxidation and promote inflammation [35], a key component of the pathogenesis of DM [36]. A targeted metabolomic approach suggested that diacyl-phosphatidylcholines C32:1, C36:1, C38:3, and C40:5 were independently associated with higher risk of DM in the EPIC Postdam cohort [37]. However, prospective data relating plasma TMAO concentration with incident DM are lacking. In particular, we did not have information on TMAO in our cohort to further explore this hypothesis. We should note that other sources of TMAO or TMAO precursors like seafood [38] could confound any egg-disease relation. In a pilot study testing 46 various foods, fish intake but not eggs or other food items led to higher urinary TMAO excretion [38]. This study further weakens the hypothesis of eggs-TMAO-DM risk. A lack of association between egg consumption and A1c, fasting glucose or insulin in our data is consistent with data from an egg feeding trial showing no effect of egg on fasting glucose in overweight men [18].

Limitations of our study include self-reported information on egg consumption and a lack of information on the size of eggs consumed, type of egg preparation, quantity of yolks consumed per egg; and on mixed dishes containing eggs. As observational study, we cannot exclude unmeasured confounding in our data. The use of an all-African American cohort limits the generalizability of our findings. However, this concern is offset by previously reported null findings in other ethnic groups. On the other hand, strengths of our study include a large sample size, availability of data on several important covariates, and use of standardized procedures to collect data in this cohort.

In conclusion, our data do not lend support for a higher incidence rate of DM with frequency of egg consumption despite a positive relation of egg consumption with prevalent DM in the JHS cohort.

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Author contribution

Conception and design of the study (LD).
Acquisition of data (PMD, HT, and KLT).
Data analysis (LD, ABP).
Interpretation of data (LD, ABP, DAH, SAT, PMD, HT, and KLT).
Drafting the article (LD) or revising it critically for important intellectual content (LD, ABP, DAH, SAT, PMD, HT, KLT).

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