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Crystal diseases

**OP0202**

**DOES EXCESS WEIGHT AFFECT GOUT RISK DIFFERENTLY AMONG GENETICALLY PREDISPOSED INDIVIDUALS? – SEX-SPECIFIC PROSPECTIVE COHORT FINDINGS OVER >26 YEARS**

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**Background:** Global burden of gout has increased substantially, particularly among women.1 Addressing obesity, a major modifiable risk factor for gout, may alleviate this burden; however, there is also a significant genetic contribution to gout risk according to the genome-wide association studies (GWAS).2,3 Genetic predisposition may modify the excess weight effect on gout.2

**Objectives:** To investigate the potential role of genetic predisposition on the association between excess weight (i.e., BMI ≥ 25 kg/m2) and gout risk in two US prospective longitudinal cohorts over >26 years, stratified by sex.

**Methods:** We examined the association between excess weight and risk of incident gout meeting the ACR survey criteria4, according to genetic risk, in 18,512 women from the Nurses’ Health Study (NHS) over 32 years, and 10,917 men from Health Professionals Follow-Up Study (HPFS) over 26 years. We derived a genetic risk score (GRS) using 114 serum urate single nucleotide polymorphisms from the latest GWAS.3 We also calculated the population attributable risk (PAR) for excess weight according to GRS stratum.

**Results:** We ascertained 530 incident gout cases in NHS and 983 in HPFS. While the relative risks (RRs) due to excess weight (overweight or obesity) appeared larger among women above the mean than below the mean, the RRs among men appeared similar according to genetic predisposition (Table 1). The RRs among women for excess weight compared to normal were 1.66 (95% CI, 1.17 to 2.37) and 2.55 (1.95 to 3.34) below and above the mean GRS, respectively (P for multiplicative interaction = 0.07). Among women with GRS above the mean (PAR, 31.6% vs 29.7%, respectively).

**Conclusion:** These large scale longitudinal prospective cohorts suggest maintaining healthy weight is an important gout prevention strategy, regardless of underlying genetic risk. In genetically predisposed individuals, addressing excess weight may prevent a large proportion of gout cases, especially among women.

**REFERENCES:**

[1] Saffi et al., PMID 32755051
[2] Xia et al., PMID 31624843
[3] Tin et al., PMID 31578528

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**Table 1. Relative Risk of Gout by Body Mass Index, Stratified by Mean Genetic Score**

<table>
<thead>
<tr>
<th>BMI</th>
<th>Overall</th>
<th>&lt;25</th>
<th>25-30</th>
<th>&gt;30</th>
<th>Overall</th>
<th>&lt;25</th>
<th>25-30</th>
<th>&gt;30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below Mean</td>
<td>Above Mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Cases</td>
<td>530</td>
<td>983</td>
<td>530</td>
<td>983</td>
<td>530</td>
<td>983</td>
<td>530</td>
<td>983</td>
</tr>
<tr>
<td>Person-Years</td>
<td>24,4392</td>
<td>12,3849</td>
<td>7,6414</td>
<td>4,4129</td>
<td>23,9259</td>
<td>12,0229</td>
<td>7,6123</td>
<td>4,2907</td>
</tr>
<tr>
<td>Age-Adjusted RR</td>
<td>1.0</td>
<td>1.06</td>
<td>1.23</td>
<td>1.46</td>
<td>3.10</td>
<td>1.23</td>
<td>1.46</td>
<td>1.80</td>
</tr>
<tr>
<td>MV Adjusted</td>
<td>1.0</td>
<td>1.00</td>
<td>1.24</td>
<td>1.48</td>
<td>3.10</td>
<td>1.24</td>
<td>1.48</td>
<td>1.80</td>
</tr>
</tbody>
</table>

*Adjusted for age (continuous), menopause, use of hormone therapy (never, past or current), history of hypertension, and systolic and diastolic blood pressure, alcohol, total energy intake and intake of meat, seafood and dairy foods (all continuous).

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**Disclosure of Interests:** None declared

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**OP0203**

**GENE-DIET INTERACTION ON THE RISK OF INCIDENT GOUT AMONG WOMEN – PROSPECTIVE COHORT STUDY OVER 32 YEARS**

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**Background:** Although gout is conventionally known as a male condition, the recent Global Burden of Disease (GBD) Study found disproportionate women’s burden over men. We have found Dietary Approaches to Stop Hypertension (DASH) diet is independently associated with a lower risk of incident gout, while Western diet is associated with increased risk.2 Whether these risks vary according to genetic risk remains unknown.

**Objectives:** To investigate the influence of genetic predisposition on the relation between diets (one protective and another hazardous) and gout risk in a large prospective US cohort of women over 32 years.

**Methods:** We examined the role of genes on the association between two dietary patterns (DASH and Western) on the risk of incident gout in 18,512 women from the Nurses’ Health Study. Using validated food frequency questionnaires, for each participant we derived: 1) DASH score emphasizing fruits, vegetables, nuts, legumes, whole grains, low-fat dairy, and reduced intake of saturated fat and sugars; and 2) Western diet score characterized by high intake of red and processed meats, SBBS, desserts, French fries, and refined grains. A genetic risk score (GRS) was derived using 114 serum urate single nucleotide polymorphisms from the latest GWAS.3

**Results:** There were 523 incident gout cases meeting ACR survey criteria4 (170 vs. 353 in GRS below and above the mean, respectively) (Table 1). Among women with GRS below and above the mean, the multivariable relative risks (RRs) of gout were 1.0, 1.56, 1.32, 0.89, and 0.61 (0.34 to 1.09) and 1.0, 1.0, 0.85, 0.51, and 0.68 (0.49 to 0.96), for quintiles (Q) 1 through 5 of DASH score, respectively (p for interaction = 0.69) (Table 1). For the Western diet, RRs for Q1 through 5 were 1.34, 1.07, 133, and 1.63 (0.91 to 2.93) for those with GRS below the mean and 1.0, 1.17, 0.93, 1.27, and 1.77 (1.19 to 2.61) among those with GRS above the mean, respectively (p for multiplicative interaction = 0.64).

**Conclusion:** In this prospective female cohort that ascertained gout with standardized criteria over 32 years, regardless of genetic predisposition, DASH diet was similarly associated with lower risk of incident gout while Western diet was associated with a higher risk. The anticipated absolute impact of diet among