GI/GL during childhood and adolescence and its relevance for metabolic outcomes - insights from observational studies

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Primary prevention of chronic disease in childhood and adolescence

Potential role of GI/GL

- Programming
  - Pregnancy
  - Early life (0-2 years)

- Shaping of long-term nutritional behaviour during childhood & adolescence

- Influence on metabolic outcomes during childhood/adolescence
  - Body weight / Body fatness
  - Risk markers of Type 2 Diabetes & CVD
    - Insulin sensitivity
    - Hepatic fat accumulation
    - Low-grade chronic inflammation
    - Serum lipids
  - Risk factors for different cancers
    - IGF-1 & IGF binding proteins
Insights from observational studies
- Outline

GI/GL during childhood and adolescence & metabolic outcomes

- Is there a link to body weight?
- Is there a link to risk markers of type 2 diabetes or CVD?

GI/GL during childhood and adolescence in every day life

- How does a lower GI/GL relate to nutrient adequacy?
- What constitutes a high dietary GI/GL among children and adolescents?
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GI/GL in DONALD

DOortmund Nutritional and Anthropometric Longitudinally Designed Study

Study - Modules

- Medical examination and anamnesis
- Anthropometry
- 3-day weighed dietary record
- 24h-urine
- Blood sample
- Parents
  - Anamnesis, Anthropometry

Age (years)

GI/GL estimation from age 1.5 years onwards
GI/GL in childhood & adolescence → body weight - observational evidence

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Study/Duration</th>
<th>Population</th>
<th>Association with GI</th>
<th>Assoc. GL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buyken 2008</td>
<td>cohort 5 years</td>
<td>380 DONALD participants (age 2y, followed-up until 7y)</td>
<td>↔</td>
<td>↔</td>
</tr>
<tr>
<td>Cheng 2009</td>
<td>cohort 4 years</td>
<td>215 DONALD adolescents (followed up during puberty)</td>
<td>↔ (↑ for overweight at puberty onset)</td>
<td>↔</td>
</tr>
<tr>
<td>Joslowski 2012</td>
<td>cohort ~5 years</td>
<td>262 DONALD adolescents (followed up until young adulthood 18-25 years)</td>
<td>↔</td>
<td>↔</td>
</tr>
<tr>
<td>Davis 2009</td>
<td>cohort 2 years</td>
<td>85 Latino-American adolescents (12-17y) with overweight</td>
<td>↔</td>
<td>↔</td>
</tr>
<tr>
<td>Gopinath 2013</td>
<td>cohort 5 years</td>
<td>856 Australian adolescents (12y followed up until 17y)</td>
<td>↔ (↑ trend ♀ BMI WC)</td>
<td>♀ BMI WC</td>
</tr>
</tbody>
</table>

↔: no association, ↑ association with higher adiposity markers

Overall evidence

Preliminary (3 cohorts), but does not support important role of GI/GL
## GI/GL in childhood & adolescence

→ body weight - intervention studies

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Study/Duration</th>
<th>Population/Intervention</th>
<th>Effect of low GI diet</th>
<th>Effect GL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spieth 2000</td>
<td>Retrosp.cohort 4 months</td>
<td>107 children &amp; adol. with obesity ad-libitum low GI diet versus kcal-red. low-fat diet</td>
<td>↓BMI, weight</td>
<td>↓BMI, fat mass</td>
</tr>
<tr>
<td>Ebbeling 2003</td>
<td>RCT 6 mo + 6 mo follow-up</td>
<td>16 adolescents with obesity ad-libitum low GL diet versus kcal-red. low-fat diet</td>
<td>↓BMI, fat mass</td>
<td>↓BMI, z-score</td>
</tr>
<tr>
<td>Parillo 2012</td>
<td>RCT 6 months</td>
<td>22 children with obesity high versus low GI diet</td>
<td>↓BMI, z-score</td>
<td>↔</td>
</tr>
<tr>
<td>Kirk 2012</td>
<td>RCT 3 mo + 6, 12 mo follow-up</td>
<td>102 children &amp; adol. with obesity Low CHO versus low GL versus standard-portion controlled</td>
<td>↔</td>
<td>↔</td>
</tr>
<tr>
<td>Mirza 2012</td>
<td>RCT 3 mo + repeated follow-up to 2 yr</td>
<td>113 Hispanic children with obesity ad-libitum low GL diet versus kcal-red. low-fat diet</td>
<td>↔</td>
<td>↔</td>
</tr>
<tr>
<td>Papadaki 2010</td>
<td>RCT 6 months</td>
<td>827 European children &amp; adol. at risk of obesity (parent) ad-lib low protein/lowGL diet vs LP/HGI vs HP/LGI vs HP/HGI vs control</td>
<td>↔</td>
<td>% overweight ↓ in HP/LGI</td>
</tr>
</tbody>
</table>

↔: equally effective as control, ↓ significantly better than control

Is combination of low GI with higher protein content more promising?
Carbohydrate nutrition in childhood & adolescence → primary prevention of overweight - overview

<table>
<thead>
<tr>
<th>Level of evidence</th>
<th>Relevant for „at risk“ individuals?</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI/GL</td>
<td>(↑)</td>
</tr>
<tr>
<td>Sugar-sweetened beverages</td>
<td>↑ OR ↑↑</td>
</tr>
<tr>
<td>(added) Sugar</td>
<td>(↑)</td>
</tr>
<tr>
<td>Dietary fibre</td>
<td>(↓)</td>
</tr>
<tr>
<td>Whole grain</td>
<td>?</td>
</tr>
</tbody>
</table>

(↑) – insufficient, some evidence suggests increased risk
↑ - possible evidence for increased risk
↑↑ - probable evidence for increases risk
(↓) – insufficient, some evidence suggests decreased risk

Limited evidence for role of GI/GL in overweight development = evidence for relevance of other aspects of carbohydrate nutrition (except sugar-sweetened beverages)
Insights from observational studies
- Outline

GI/GL during childhood and adolescence & metabolic outcomes

- Is there a link to body weight?
- Is there a link to risk markers of type 2 diabetes or CVD?

GI/GL during childhood and adolescence in every day life

- How does a lower GI/GL relate to nutrient adequacy?
- What constitutes a high dietary GI/GL among children and adolescents?
Critical periods for risk of chronic diseases

Potentially influencing factors

- Excessive pregnancy weight gain
- Maternal overweight
- Gestational diabetes
- Birth weight
- Formula feeding
- Rapid weight gain (0-2 years)
- High GI?

Critical Periods

- Conception
- Birth
- 2
- 4
- 6
- 8
- 10
- 12
- 14
- 16
- 18

Periods of physiological insulin resistance

- prenatal
- Early childhood
- Adiposity rebound
- Puberty

High GI?
Puberty as a potentially sensitive window for GI/GL

- **Physiological IR** (Goran et al. 2003)
  - decrease in peripheral insulin sensitivity
  - higher levels of fasting glucose and insulin
  - increase in acute insulin response is disproportionately low

- **Other hormonal changes**, i.e. regulators of
  - satiety, appetite
  - body fat distribution

- **Behavioural changes** (Alberga et al. 2012)
  - decreases in physical activity
  - increases in sedentary activities (TV, computer etc.)
  - dietary changes
    - lower intakes of fruits and vegetables
    - increased consumption of fast foods / soft drinks
    - lower participation in family meals, more eating outside home
GI/GL in childhood and adolescence → risk markers of type 2 diabetes or CVD observational evidence

Cross-sectional studies

- GL → HDL ↓ in adolescents (Slyper et al. 2005)
- GL → risk of metabolic syndrome ↑ in adolescents (O'Sullivan et al. 2010)
- GI, GL: no association with insulin dynamics in adolescents (Davis et al. 2007)
- GI → narrower retinal arterioles in adolescent girls (Gopinath et al. 2012)

Prospective studies

- GI & GL → systolic blood pressure ↑ in adolescent girls (Gopinath 2012)
DONALD Study: GI/GL in puberty

**DO**rtmund **Nutritional and Anthropometric Longitudinally Designed Study**

- Anthropometric data at the beginning of puberty
- Information about potential confounder
- Term singletons

**Study sample:**

n = 226

At least 2 plausible 3-day weighed dietary records (♀ 9-14 y, ♂ 10-15 y)

Blood data in young adulthood (18-36 y)
### Tertiles of dietary GI during puberty

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean GI in tertiles</td>
<td>53.4</td>
<td>56.1</td>
<td>58.4</td>
<td></td>
</tr>
<tr>
<td>Energy (MJ/day)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>8.0</td>
<td>7.6</td>
<td>8.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Fat (% energy)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>35.4</td>
<td>36.5</td>
<td>35.4</td>
<td>0.2</td>
</tr>
<tr>
<td>Protein (% energy)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>13.4</td>
<td>13.0</td>
<td>12.5</td>
<td>0.0007</td>
</tr>
<tr>
<td>Carbohydrate (% energy)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>51.1</td>
<td>50.5</td>
<td>52.1</td>
<td>0.08</td>
</tr>
<tr>
<td>Added sugar (% energy)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>13.1</td>
<td>14.2</td>
<td>17.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Fibre (g)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>20.6</td>
<td>18.9</td>
<td>17.3</td>
<td>0.0005</td>
</tr>
<tr>
<td>Whole grain (g)&lt;sup&gt;2&lt;/sup&gt;</td>
<td>29.1</td>
<td>19.2</td>
<td>16.3</td>
<td>0.0004</td>
</tr>
</tbody>
</table>

<sup>1</sup> Mean  
<sup>2</sup> Median
GI in puberty
↔ Insulin resistance (HOMA-IR) in young adulthood

n=226 DONALD participants
at least two 3-day weighed dietary records (average 5) in puberty
→ risk markers of type 2 diabetes in younger adulthood (18-35 years)

Adjusted for sex, age, firstborn status, BMI SDs during puberty, maternal education, energy, protein and fibre intake.

No associations with total carbohydrates, dietary GL, total added sugar, dietary fiber, or whole-grain intake.

Goletzke et al. Diabetes Care 2013
GI in puberty ↔ markers of hepatic steatosis in young adulthood

n=214 DONALD participants

Adjusted for sex, age, BMI SDs during puberty, maternal overweight, energy, protein and fibre intake.

No associations with total carbohydrates, dietary GL, total added sugar, dietary fiber, or whole-grain intake.

Goletzke et al. Diabetes Care 2013
Carbohydrates and GL in puberty ↔ chronic inflammation in young adulthood

n=205 DONALD participants

Total carbohydrates (en%)

Glycaemic Load

adjusted for sex, age, BMI-SDs during puberty, gestational weight gain, maternal overweight, energy and whole grains

no associations with hs-CRP, adiponectin or IL-18

Goletzke et al. in preparation
Carbohydrates from high and low GI sources in puberty ↔ IL-6 in young adulthood

n=205 DONALD participants

Carbohydrates (en%) from foods with GI > 55

Carbohydrates (en%) from foods with GI ≤ 55

A higher dietary GI and carbohydrates from high GI food sources consumed during puberty may affect type 2 diabetes risk in later life

adjusted for sex, age, BMI-SDs during puberty, gestational weight gain, maternal overweight, energy and whole grains
GI in puberty ↔ other markers in young adulthood

n=213 DONALD participants

IGF-I

\[ P\text{-trend} = 0.3 \]

IGFBP-3

\[ P\text{-trend} > 0.9 \]

Also, no prospective associations GI/GL with triglycerides or HDL cholesterol in young adulthood

Tertiles of dietary GI during puberty

adjusted for age, maternal education, smokers in the household, fat free mass index during puberty, protein intake

unpublished data
Insights from observational studies
- Outline

GI/GL during childhood and adolescence & metabolic outcomes

- Is there a link to body weight?
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GI/GL during childhood and adolescence in every day life

- How does a lower GI/GL relate to nutrient adequacy?
- What constitutes a high dietary GI/GL among children and adolescents?
Carbohydrates from high GI sources in Australian children and adolescents
↔ nutrient adequacy - ANCNPS 2007 (n=4140)

<table>
<thead>
<tr>
<th>Quartiles of carbs from high GI foods (GI&gt;52)</th>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median CHO (g)</td>
<td>90</td>
<td>110</td>
<td>134</td>
<td>189</td>
<td></td>
</tr>
<tr>
<td>Iodine¹</td>
<td>1.0</td>
<td>2.13</td>
<td>2.71</td>
<td>5.45</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Calcium¹</td>
<td>1.0</td>
<td>1.13</td>
<td>1.71</td>
<td>3.13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Vitamin A RE¹</td>
<td>1.0</td>
<td>1.31</td>
<td>2.19</td>
<td>3.77</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LC n-3 PUFA¹</td>
<td>1.0</td>
<td>1.30</td>
<td>1.21</td>
<td>1.81</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

¹ adjusted for age, sex and total energy intake

Louie et al. AJCN 2012

Also, increased risk for not meeting NRVs for:
Potassium, Phosphorus, Zinc, Riboflavin, Vitamin D, Vitamin E
Carbohydrates from high GI sources in Australian children and adolescents ↔ nutrient adequacy - ANCNPS 2007 (n=4140)

### Odds ratios for not meeting Australian reference values

<table>
<thead>
<tr>
<th>Quartiles of carbs from low GI foods (GI≤52)</th>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median CHO (g)</td>
<td>71</td>
<td>91</td>
<td>114</td>
<td>160</td>
<td></td>
</tr>
<tr>
<td>Iodine&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.0</td>
<td>0.44</td>
<td>0.34</td>
<td>0.24</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Calcium&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.0</td>
<td>0.58</td>
<td>0.44</td>
<td>0.35</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Vitamin A RE&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.0</td>
<td>0.68</td>
<td>0.48</td>
<td>0.43</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LC n-3 PUFA&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.0</td>
<td>1.51</td>
<td>2.00</td>
<td>2.31</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

<sup>1</sup> adjusted for age, sex and total energy intake

Louie et al. AJCN 2012

Also, decreased risk for not meeting NRVs for:
- dietary fibre
- potassium
- magnesium
- folate
- Vitamin C

Also, increased risk for not meeting NRVs for LA and ALA

Higher consumption of carbohydrates from low GI sources confers benefits for overall nutrient adequacy
Insights from observational studies
- Outline

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GI/GL during childhood and adolescence in every day life

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GI in Australian children and adolescents ↔ relation to carbohydrate quality
ANCNPS 2007 (n=4140)

Pearson’s correlation

<table>
<thead>
<tr>
<th></th>
<th>2-3y</th>
<th>4-8y</th>
<th>9-13y</th>
<th>14-16y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sugars (%energy)(^1)</td>
<td>-0.16*</td>
<td>-0.20*</td>
<td>-0.16*</td>
<td>-0.10*</td>
</tr>
<tr>
<td>Starch (%energy)(^1)</td>
<td>0.44*</td>
<td>0.41*</td>
<td>0.34*</td>
<td>0.34*</td>
</tr>
<tr>
<td>Fibre density (g/MJ)(^1)</td>
<td>0.02</td>
<td>-0.09</td>
<td>-0.08*</td>
<td>-0.14*</td>
</tr>
</tbody>
</table>

\(^1\) adjusted for age, sex and total energy intake
*significant at p<0.05

Louie et al. BJN 2011

A higher GI is associated with a higher starch intake
Associations with a lower sugar intake and a lower fibre density are modest

Current advice to increase consumption of dietary fibre/whole grains and/or reduce consumption of sugary foods cannot be expected to translate into a reduction of dietary GI
Carbohydrate sources contributing to GL in paediatric populations

Main contributors to GL in German DONALD participants aged 7-8 years
(Buyken et al. 2002)

1. Tolerated food groups (30%) (sweets, soft drinks, cakes and cookies, and salty snacks)
2. Bread and rolls (25%)
3. Milk and milk products (10%)
4. Breakfast cereals and cereals (7%)
5. Fruits (7%)

Main contributors to GL in Australian children and adolescents ANCNPAS 2007
(Louie et al. 2011)

2-3 years
1. Fruit (11%)
2. Breakfast cereals (11%)
3. White bread (9%)
4. Biscuits (7%)
5. Cows’ milk (unflavoured) 6%

Energy-dense and/or nutrient poor food groups ~ 29%

14-16 years
1. White bread (12%)
2. Breakfast cereals (9%)
3. Cakes, pastries & doughnuts (8%)
4. Soft drinks (7%)
5. Fast food (6%)

Energy-dense and/or nutrient poor food groups ~ 42%
Summary

GI/GL during childhood and adolescence & metabolic outcomes

- Current evidence does not support a major role of dietary GI/GL in the development of overweight and obesity in childhood or adolescence.

- A higher dietary GI and carbohydrates from high GI food sources consumed during puberty may affect type 2 diabetes risk in later life.

- Hence, advice for preferred selection of low-GI carbohydrates during puberty should be incorporated into preventive dietary recommendations given to adolescents.

- Consideration of GI/GL already in childhood may also be warranted because long-term nutritional behaviours are shaped early in life.
Summary

GI/GL during childhood and adolescence in every day life

- Preferred selection of carbohydrates from low GI sources may confer benefits for overall nutrient adequacy

- Current advice to increase consumption of dietary fibre/whole grains and/or reduce consumption of sugary foods cannot be expected to translate into a reduction of dietary GI – dietary GI should be considered as an additional entity

- Efforts to reduce dietary GI and GL in children and adolescents should focus on energy-dense starchy foods

- Advice regarding replacement of high GI carbohydrates for lower GI alternatives should consider local carbohydrate intake patterns
Acknowledgements

- **Participants** of the DONALD Study and their parents
- **Collaborators**
  - Prof. Dr. Thomas Remer, Dortmund
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  - Dr. Jimmy Louie & Dr. Victoria Flood, Sydney
  - Dr. Christian Herder & Prof. Michael Roden, Düsseldorf
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  - Gesa Joslowski
  - Janina Goletzke
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