**WORKSHEET: Etiology/Harm**

**Citation:** McGregor SE, Courneya KS, Kopciuk KA, Tosevski C, Friedenreich CM. Case–control study of lifetime alcohol intake and prostate cancer risk. Cancer Causes Control (2013) 24:451–461

<table>
<thead>
<tr>
<th>1. Are the Results Valid?</th>
<th>[For CCS] Were cases and controls similar with respect to the indication or circumstances that would lead to exposure?</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Yes</strong> No Unsure</td>
<td><img src="#" alt="Table content" /></td>
</tr>
<tr>
<td><strong>Yes</strong> No Unsure</td>
<td>[For CCS] Were the circumstances and methods for determining exposure similar for cases and controls?</td>
</tr>
</tbody>
</table>

**Name:**

**Citation:** McGregor SE, Courneya KS, Kopciuk KA, Tosevski C, Friedenreich CM. Case–control study of lifetime alcohol intake and prostate cancer risk. Cancer Causes Control (2013) 24:451–461
<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
<th>Unsure</th>
<th>Do the results satisfy some “diagnostic tests for causation”? (see Causal Criteria page below)</th>
</tr>
</thead>
</table>

**Rate the validity of the study [circle your ranking].**

- Extensive Flaws
- Major Flaws
- Minor Flaws
- Minimal Flaws

**2. What are the results?**

- How strong is the association between exposure and outcome (RR’s or OR’s)?

- How precise is the estimate of the risk (CI’s)?
What is the magnitude of the risk associated with lifetime alcohol intake? Calculate the lifetime NNH for dying of prostate cancer in patients who consume on average 21 drinks of alcohol per week or more compared to non-drinkers. Use the lifetime risk of dying of prostate cancer = 2.7%. as reported by the ACS using SEER data (Ref: [http://www.cancer.org/cancer/cancerbasics/lifetime-probability-of-developing-or-dying-from-cancer](http://www.cancer.org/cancer/cancerbasics/lifetime-probability-of-developing-or-dying-from-cancer)).

Use the formula for deriving NNH from ORs (see Table 10.2-7, page 227 of Users’ Guide).

<table>
<thead>
<tr>
<th>3. How can I apply the results to my patient care?</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Yes</strong></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
<th>Unsure</th>
<th>Is the exposure similar to what might occur in my patient? (are there important differences in the dose, or duration of exposure?)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>Unsure</td>
<td>Are there any benefits that offset the risk associated with exposure?</td>
</tr>
<tr>
<td>Yes</td>
<td>No</td>
<td>Unsure</td>
<td>Should I attempt to stop the exposure?</td>
</tr>
</tbody>
</table>
The Causal Criteria:
Below is a list of causal criteria that we use to help determine cause and effect. We will
review these in more detail in the last week of the class. However, read the following
descriptions and try to answer each criterion based on the information provided in the
CCS:

1. **Strength of association** - a large effect (e.g., RR > 5) is less likely to result from
   confounding or bias.

2. **Time Order** – did the exposure precede the disease?

3. **Dose-effect** – does risk increase with greater exposure (i.e., amount and/or
duration)?

4. **Specificity** – Is the effect of an exposure specific – meaning, does it only cause one
effect?

5. **Plausibility** - is a cause effect relationship plausible on biologic grounds?

6. **Consistency** - Finding the effect in more than one study, population, study design or
   sub-group.
   a. **Internal consistency** - Within the study does the effect persists among different
   sub-groups?
   b. **External consistency** – Have other investigators found similar findings?

7. **Reversibility** – Does removal of a presumed cause lead to a reduction in the risk of ill-
   health?

8. **Analogy** - Are there other established cause and effect relationships that have a
   similar exposure or disease?
Optional questions about this study:

1. List several possible reasons why some case-control studies of lifetime alcohol intake did NOT find an association with prostate cancer risk (see discussion, pg 457)

2. How did investigators minimize the different types of bias:

   Selection bias –

   Confounding bias –

   Recall bias –

   Measurement bias –

3. What characteristic of prostate cancer makes the disease susceptible to environmental factors such as diet and alcohol use?

4. Why was a sensitivity analysis performed using the lowest levels of alcohol intake as the reference group? (Methods, page 453)

5. A larger proportion of controls had a higher education level than cases; how might this characteristic affect prostate cancer risk?