Effects of Acute Cold Exposure on Cognitive Function: Evidence for Sustained Impairment

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INTRODUCTION

The body undergoes numerous physiological changes in response to cold temperatures in an attempt to maintain thermoregulatory homeostasis. Shivering thermogenesis, modified circulatory patterns, and increased catecholamine production serve to increase heat production and minimize heat loss. Interestingly, cerebral changes also occur in response to cold temperatures, including changes in cerebral perfusion, disruption of the blood brain barrier, and increased catecholamine expression (Makinen et al., 2006; Wagner & Zuccarello, 2005, Schmoker et al., 2009).

Given these neurological changes, it is not surprising cold exposure is also associated with reduced cognitive functioning. Although methodological differences across studies make direct comparison difficult (e.g. severity of cold, specific cognitive tests used), there is reason to believe that performance decreases on speeded cognitive tasks and mildly improves on many others in response to moderate cooling (Palinkas et al., 2005; Makinen et al., 2006). Although the exact mechanisms for cold-related cognitive dysfunction remain unknown, researchers have suggested that distraction might serve to interfere with speeded tasks, whereas the greater physiological arousal resulting from cold exposure could improve performance on untimed, more complicated tasks (Teichner, 1958; others here).

However, past studies have typically examined cognitive function only prior to and during cold exposure. This methodology fails to capitalize on the drop in core temperature observed after removal from cold environments, often termed after-drop (Collins, 1983). Serial administration of cognitive tests before, during, and after cold exposure will help to clarify the underlying mechanisms. The present study did so in a sample of healthy young adult males. Testing was completed prior to cold exposure, during cold exposure, approximately 60 minutes after removal from cold, and four hours after removal from cold.

METHODS

The purpose of the investigation was to examine the effects of acute cold exposure (ACE) on cognitive function. Data for the current study are part of a larger project examining the interactive effects of sleep deprivation and ACE on physiological functioning. Only information from the control condition (i.e. no sleep deprivation) and relevant to the current hypotheses are presented below.

Each participant visited the Kent State University Exercise Physiology Laboratory on two separate occasions. During the first visit, baseline measures of demographic variables, medical
characteristics, and cognitive function was obtained. During the second visit, participants underwent serial ACE and cognitive assessment over a three-day period.

*Participants*

The volunteers for this study were 10 apparently healthy college-aged Caucasian males. Prior to the commencement of data collection, approval from the Kent State University Institutional Review Board was obtained. All participants were required to complete a general medical history questionnaire and sign a letter of informed consent prior to participation in the study. All participants were apparently healthy, with no known metabolic disorders, free of disease and currently not taking any medications that could affect thermoregulation, metabolism, hormonal or cognitive responses. All participants were within the normal body composition percentiles (i.e., 30th – 70th percentile) and normal fitness ranges for age for cardiovascular endurance (30th – 70th percentile for age) as stated by the American College of Sports Medicine.

*Procedures*

Participants reported to the laboratory at 9:00pm prior to the start of the trial. Participants were asked to go to sleep at 10:00pm in a supervised laboratory and were woken up at 5:00am to undergo the first ACE at 6:00-8:00am. During ACE participants were exposed to 10°C air for 120 minutes beginning at 6:00am until 8:00am. Temperature within the chamber was monitored to ensure control within 0.5°C of 10°C. Participants were dressed in athletic shorts, gloves and socks for the trial. Immediately following ACE, re-warming (REC-1) was examined as participants remained in the environmental chamber for 120 minutes in 25°C air.

During the initial stage of recovery (REC-1) the participant remained seated quietly in a lounge chair in 25°C. During ACE and REC-1, core and skin temperatures were monitored continuously. REC-2 was completed approximately four hours later in 25°C temperatures after changing into street clothes. Participants were asked to return to sleep at 10:00pm in the laboratory. The participant was woken up at 5:00am to complete a second ACE followed by REC-1 and REC-2. The participant again remained supervised in the laboratory for the remainder of the day and was asked to return to bed at 10:00pm. Participants were awoken at 5:00am to complete a third and final ACE and REC-1 and REC-2. After the completion of the third ACE-REC, participants were released to return home.

Throughout the study period, participants were fed a standardized diet and blood glucose was monitored to ensure euglycemia.

*Measures*

**Temperature**

Core temperature (°C) was measured continuously throughout baseline, ACE, and REC-1 using a rectal thermometer (ER 400-12, O.E. Meyer Co., Sandusky, Ohio). The participant was instructed to insert the rectal thermometer 13cm beyond the anal sphincter. Skin temperature (°C) was measured continuously throughout baseline and the ACE with the use of skin thermistors (Model 409B, Yellow Springs Instruments Inc., Dayton, Ohio). The thermistors were applied to the right chest, tricep, forearm, thigh and calf of the participant’s body (Toner et al., 1986) and held in place by waterproof tape (Hy-tape® International, Patterson, New York). Following completion of the ACE, both rectal and skin thermistors were removed until the next bout of cold exposure. Mean skin temperature was calculated using the formula utilized by Toner, Garfinkel, and Garner (1986): \( T_{sk} = (0.22 \times \text{calf temperature}) + (0.28 \times \text{thigh temperature}) + (0.28 \times \text{chest temperature}) + (0.14 \times \text{forearm temperature}) + (0.08 \times \text{tricep temperature}) \)
Core temperature ($T_{core}$) and skin temperature ($T_{sk}$) data were then gathered via an interface (iNet-100HC, Omega Engineering, Inc., Stamford, Connecticut) to a PC and recorded every minute.

**Thermal Sensation**

Thermal sensation (TS) was measured using the Gagge Thermal Sensation Scale (Gagge, Stolwijk, & Hardy, 1967). The Gagge Thermal Sensation Scale is a valid and reliable measure of subjective whole body thermal sensation. Participants were asked to measure TS at during BASE and at 5, 15, 30, 45, 60, 75, 90, 105, and 120 minutes during ACE and REC-1.

**Cognitive Tests**

A computerized test battery was administered to participants at each of the four time points, PRE-ACE, ACE, REC-1 (average of 60 minutes post-ACE), and REC-2 (average of 4 hours post-ACE). The test battery is both reliable and valid (Paul et al., 2005; Williams et al., 2005). The abbreviated test battery required 20 minutes complete and all scoring of cognitive tests are done at a central facility using an automated software program for most tests and hand-scoring for wav files.

Specific tests included in this study included:

a. Attention:

Digit Span: This test assesses basic auditory attention and working memory. Participants are presented with a series of digits on the touch-screen, separated by a one-second interval. The subject is then immediately asked to enter the digits on a numeric keypad on the touch-screen. In the first part of the test, subjects are required to recall the digits in forward order and reverse order in the second. In each part, the number of digits in each sequence is gradually increased from 3 to 9, with two sequences at each level. The dependent measure is the total number of correct trials forward and backward.

Choice Reaction Time: This test assesses choice reaction time and sustained performance. Participants attended to the computer screen as one of four circles was illuminated. Immediately following presentation, the subject then had to touch the illuminated circle as quickly as possible. Twenty trials were administered with a random delay between trials of 2-4 seconds. Dependent variables include mean reaction time and the standard deviation of response time.

b. Executive Function:

Verbal Interference: This task taps the ability to inhibit automatic and irrelevant responses and has similarities to the Stroop task (Golden, 1978). In Verbal Interference Part 1, all words are printed in black ink and participants are asked to read each word as quickly as possible (e.g. “blue” printed in black ink, “red” printed in black ink). In Verbal Interference part 2, words are printed in incongruent colors (e.g. “red” printed in blue ink, “green” printed in red ink). Total number of words correctly read served as the dependent variables for analyses.

Executive Maze Task: This task is a computerized adaptation of the Austin Maze (Walsh, 1991). Participants are presented with a grid (8x8 matrix) of circles and asked to identify the hidden path through the grid. Distinct auditory and visual cues are presented for correct and incorrect responses. The trial ends when the subject completed the maze twice without error or after 10 minutes has elapsed. The dependent variables include the number of maze errors and maze overruns.

**Data Analysis**
Generalized estimating equations (GEE) for panel data in Stata v10 were used to examine cognitive function. GEE analyses all data at all timepoints simultaneously. All positively skewed outcomes were log-transformed prior to analysis, but plots show raw scores. Any outcome observations that were extreme outliers were excluded on analysis-by-analysis basis. Measures of cold (i.e. core temperature, skin temperature, and reported thermal sensation) were analyzed using repeated measures ANOVA for each of the 20 time points.

RESULTS

GEE analyses showed that exposure to cold temperatures impacted performance on multiple measures of cognitive function, including Digit Span, Choice Reaction Time, Verbal Interference Part 1, and Verbal Interference Part 2. No effect was found for Executive Maze test performance. See Table 1 below. For each of these tests, performance at ACE and REC-1 was poorer than PRE-ACE and REC-2.

Table 1
Cognitive Test Scores

<table>
<thead>
<tr>
<th>Measure</th>
<th>PRE-ACE</th>
<th>ACE</th>
<th>REC-1</th>
<th>REC-2</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Digit Span</em></td>
<td>7.38±1.36</td>
<td>7.08±2.51</td>
<td>7.19±2.28</td>
<td>7.88±2.97</td>
</tr>
<tr>
<td><em>Choice React.</em></td>
<td>630.30±97.74</td>
<td>690.32±116.20</td>
<td>728.40±124.25</td>
<td>649.10±106.24</td>
</tr>
<tr>
<td>*Verbal Part 2</td>
<td>17.27±4.22</td>
<td>19.46±2.89</td>
<td>18.77±3.04</td>
<td>21.42±3.11</td>
</tr>
<tr>
<td>Maze Error Overrun</td>
<td>12.27±8.98</td>
<td>14.62±8.07</td>
<td>12.42±6.07</td>
<td>17.23±17.78</td>
</tr>
</tbody>
</table>

Note: Mean± Standard Deviation. * denotes significantly lower performance at ACE and REC-1 than baseline and REC-2.

In terms of response to the cold temperatures, repeated measures ANOVA indicated a significant change in core temperature \(\lambda=0.02, F(19, 10)=24.99, p<.001\), skin temperature \(\lambda=0.01, F(19, 4)=86.33, p<.001\), and reported thermal sensation \(\lambda=0.03, F(19, 8)=16.02, p<.001\). See Table 2 below. Core temperatures increased during ACE and slowly returned to original levels at the end of the recovery period. Skin temperature and thermal sensation showed a significant response to ACE, followed by a more rapid return to baseline levels.

Table 2.
Core temperature, skin temperature, and thermal sensation in 10 healthy young adults

<table>
<thead>
<tr>
<th>Timepoint</th>
<th>Core Temperature</th>
<th>Skin Temperature</th>
<th>Thermal Sensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-ACE</td>
<td>36.65 ± 0.24</td>
<td>31.06 ± 0.30</td>
<td>4.05 ± 0.59</td>
</tr>
<tr>
<td>ACE 5min</td>
<td>36.85 ± 0.22</td>
<td>25.35 ± 1.33</td>
<td>2.58 ± 0.92</td>
</tr>
<tr>
<td>ACE 30 min</td>
<td>37.02 ± 0.21</td>
<td>23.07 ± 1.38</td>
<td>2.30 ± 0.84</td>
</tr>
<tr>
<td>ACE 60 min</td>
<td>37.00 ± 0.24</td>
<td>22.39 ± 1.41</td>
<td>1.97 ± 0.83</td>
</tr>
<tr>
<td>ACE 90 min</td>
<td>36.94 ± 0.22</td>
<td>21.95 ± 1.71</td>
<td>1.67 ± 0.77</td>
</tr>
<tr>
<td>ACE 120 min</td>
<td>36.86 ± 0.24</td>
<td>21.76 ± 1.58</td>
<td>1.62 ± 0.86</td>
</tr>
<tr>
<td>REC 5 min</td>
<td>36.84 ± 0.24</td>
<td>22.71 ± 1.41</td>
<td>2.43 ± 0.79</td>
</tr>
<tr>
<td>REC 30 min</td>
<td>36.71 ± 0.26</td>
<td>26.69 ± 1.24</td>
<td>3.40 ± 0.54</td>
</tr>
<tr>
<td>REC 60 min</td>
<td>36.56 ± 0.23</td>
<td>28.19 ± 0.94</td>
<td>3.78 ± 0.59</td>
</tr>
<tr>
<td>REC 90 min</td>
<td>36.53 ± 0.22</td>
<td>28.61 ± 0.80</td>
<td>3.81 ± 0.52</td>
</tr>
<tr>
<td>REC 120 min</td>
<td>36.54 ± 0.22</td>
<td>28.68 ± 0.86</td>
<td>3.91 ± 0.86</td>
</tr>
</tbody>
</table>
CONCLUSIONS

Results from the current study indicate that cognitive function is reduced both during acute exposure to cold temperatures and for a short period of time afterward. This pattern emerged despite multiple measures of cold (e.g. core/skin temperatures, thermal sensation) being similar to baseline levels. Such findings suggest that the physiological adaptations to maintain thermoregulatory homeostasis adversely impact cognitive function, though substantial future work is needed to clarify the exact mechanisms. Researchers are particularly encouraged to employ non-invasive measures of cerebral blood flow and circulating biomarkers.

Several methodological considerations may limit generalizability to other samples. As the cognitive impact of cold temperatures remains poorly understood, the most appropriate temperature and duration of cold exposure for examination has not been established. It is possible that lower temperatures or a long duration may produce different findings. Additionally, there is reason to believe that thermoregulation differs by racial/ethnic groups (Farnell et al 2008) and gender (Glickman-Weiss et al, 2000). and little is known about the impact of these moderator variables on cognitive function in the cold.

REFERENCES