

## Purpose

To investigate the NoGo N2 and P3 in a sample of violent offenders with a full range of psychopathy and non-offender controls.

## Background

### Anterior Cingulate Cortex (ACC):

Heavily implicated in response monitoring and control.<sup>1</sup>

Considered the generator of the NoGo N2<sup>1</sup>

Reciprocal connections with areas that generate NoGo P3<sup>2</sup>

### Psychopathy:

Some reports of diminished inhibitory control under some conditions.<sup>3</sup>

Evidence suggesting abnormal ACC function during some processing conditions.<sup>4,5</sup>

Some evidence of abnormal response monitoring during a Go/NoGo task<sup>6</sup>

*Is there evidence of a diminished NoGo N2/P3 in violent offenders diagnosed with psychopathy?*

## Methods

### Participants

- 14 violent offenders (Mean age = 47.5)
  - Mean PCL-R score = 25.8
- 14 control participants (Mean age = 46.15 years)

### Procedure

- Serial presentation of a series of letters (e.g. X, Y) in alternating fashion
  - **Go Condition:** Current letter was different from the preceding one
  - **NoGo Condition:** Current letter is the same as on the preceding trial
- 550 stimuli presented + 30 practice trials
- 1/3 trials NoGo

### ERPs

- 128 site Biosemi system
- 512 points per second
- Baseline: 200ms prestimulus
  - offline filtered 1 to 30 Hz

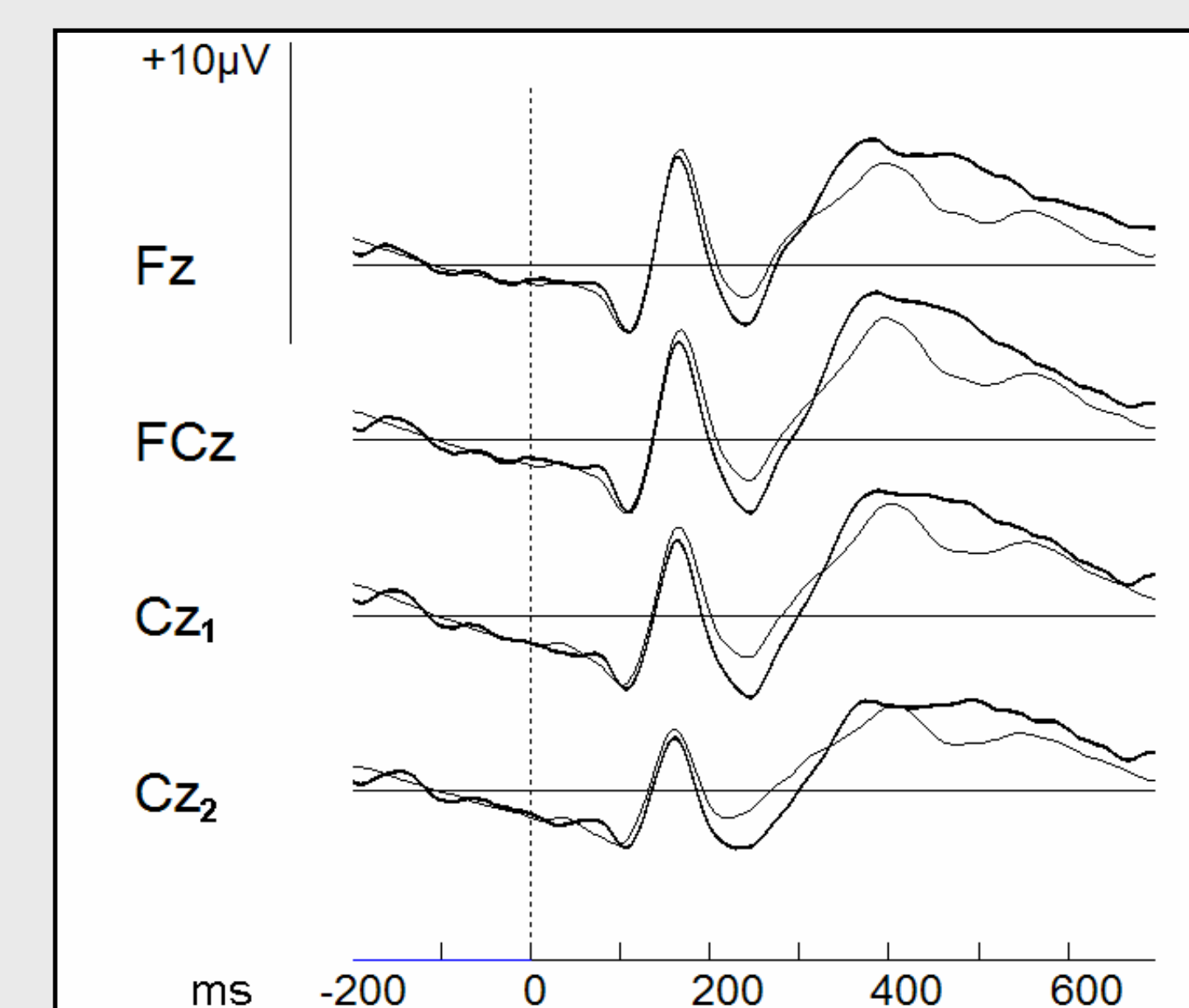
## Results

• Offenders ( $M = 46 \pm .04$ ) had more errors of commission on NoGo trials than Controls ( $M = .32 \pm .03$ ),  $p = .035$

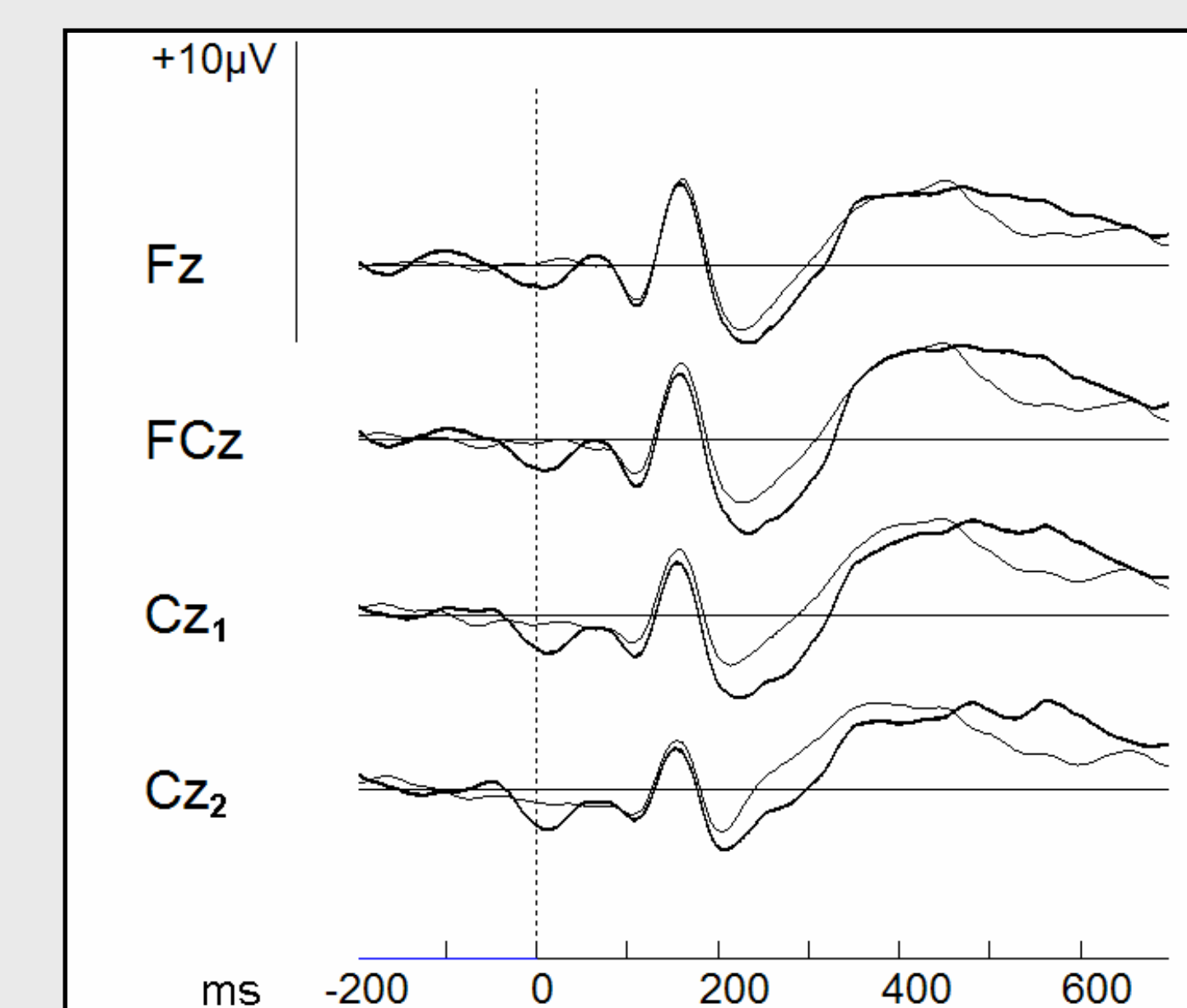
- This was unrelated to psychopathy
- Both groups showed a strong NoGo N2 amplitude effect  $p < .001$ 
  - No Group or Latency effects
- Offenders generated smaller P3s at frontal sites  $p = .023$ 
  - No relation between psychopathy and P3 amplitude or latency

## ERPs by Group

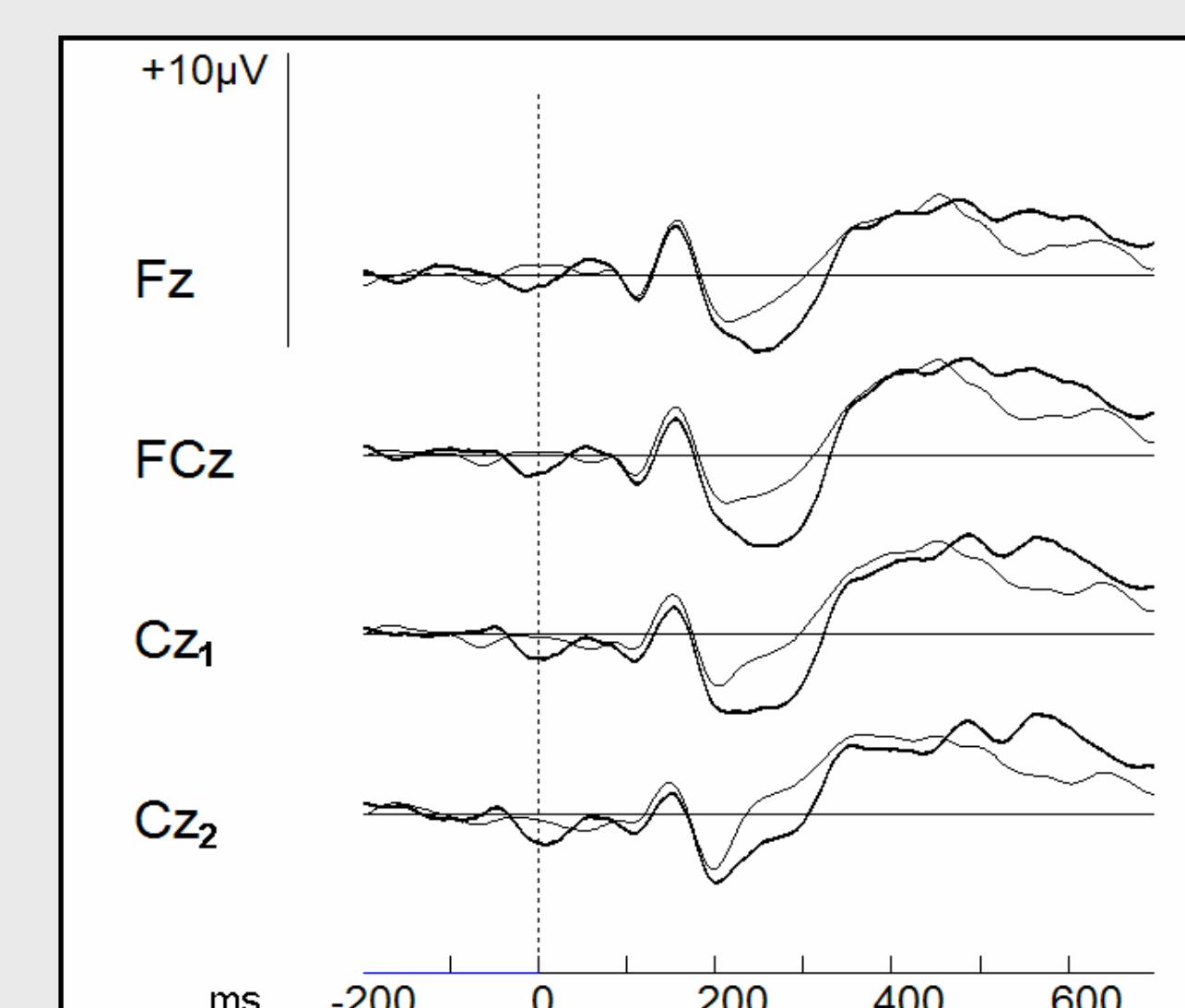
### A. Control Group



### B. Offender Group



### C. Psychopath Subgroup PCL-R = 31.78



### D. Nonpsychopath Subgroup PCL-R = 15.0

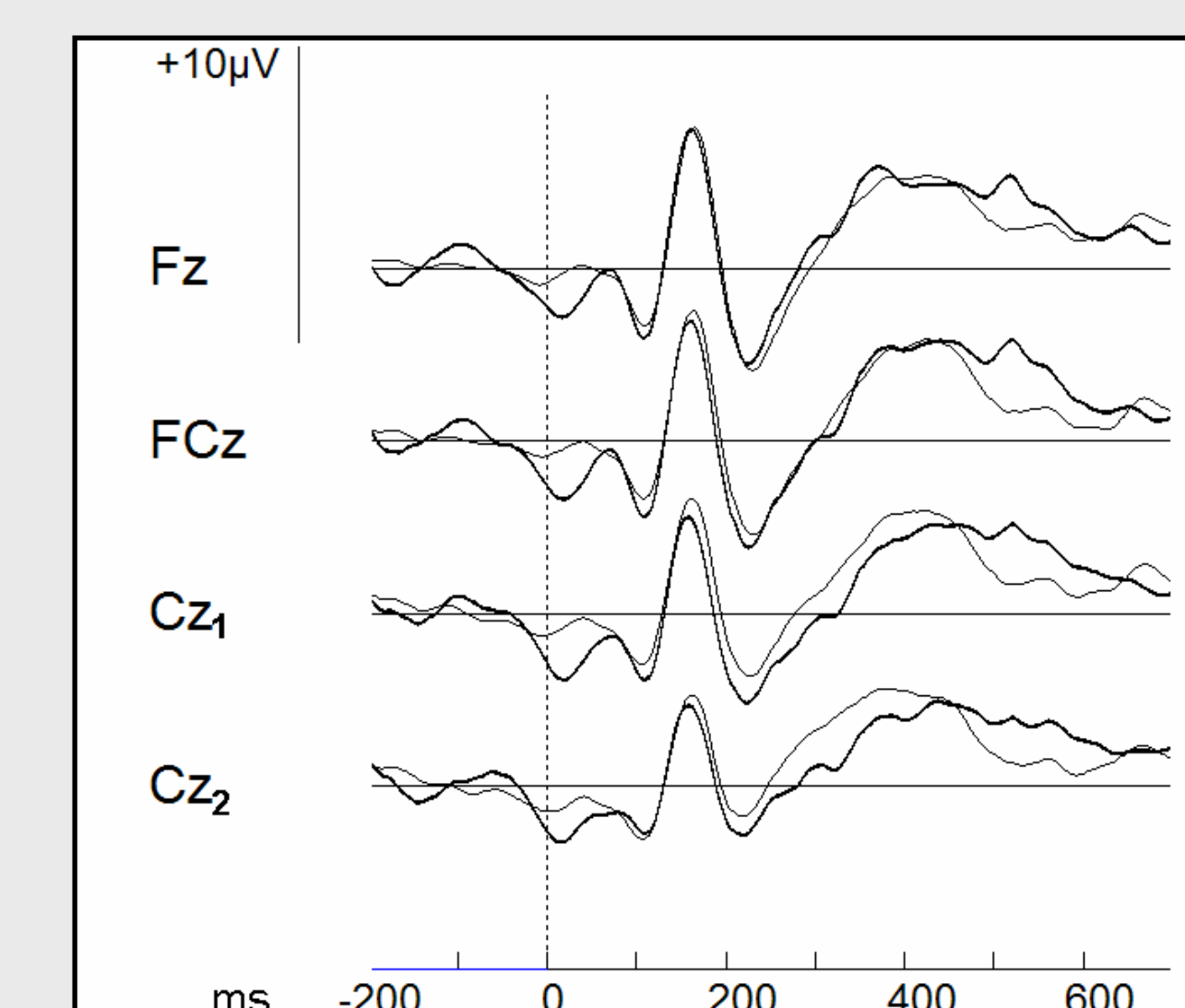


Table 1.

Amplitude and latency data for the N2 and P3 by electrode site and group.

	ERP Component			
	N2 amplitude ( $\mu V$ )	N2 latency (ms)	P3 amplitude ( $\mu V$ )	P3 latency (ms)
Controls				
Fz	-1.81	250	5.34	443
FCz	-2.24	254	6.20	439
Cz1	-2.76	254	5.84	470
Cz2	-2.59	255	4.85	486
<i>M</i>	-2.35	253	5.56	459
<i>(SE)</i>	(.77)	(12.11)	(.63)	(23.01)
Offenders				
Fz	-3.50	231	4.29	488
FCz	-4.02	234	5.01	502
Cz1	-4.07	231	5.09	469
Cz2	-3.29	232	4.96	472
<i>M</i>	-3.72	232	4.83	483
<i>(SE)</i>	(.77)	(12.11)	(.63)	(23.01)

Table 2.

Behavioral data by group.

	Reaction Times (ms)		Proportion of Correct Hits		Errors of Commission	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
Controls	364	13.8	.89	.01	.32	.03
Offenders	418	14.4	.90	.01	.46	.04
Psychopaths	410	21.5	.88	.02	.43	.04
Nonpsychopaths	435	32.9	.94	.03	.52	.06

## Conclusions

Psychopathy was not associated with atypical behavioral response or diminished frontal ERPs under these testing conditions.

Abnormal function of ACC and associated regions may only occur with emotional stimuli<sup>5,7</sup> or under testing conditions involving affectively-charged feedback contingencies.<sup>8</sup>

## References

1. Ridderinkhof, K.R., Ullsperger, M., Crone, E.A. & Nieuwenhuis, S. (2004). *Science*, 306, 443-447.
2. Bokura, H., Yamaguchi, S., & Kobayashi, S. (2001). *Clin. Neurophysiology*, 112, 2224-2232.
3. Newman, J.P., & Smith, W.A. (1998). *Journal of Abnormal Psychology*, 107, 527-532.
4. Kiehl, K. A. et al. (2001). *Biol. Psychiatry*, 50, 677-684.
5. Muller, J. et al. (2003). *Biol. Psychiatry*, 54, 152-162.
6. Kiehl, K.A., Smith, A.M., Hare, R.D., & Liddle, P.F. (2000). *Biol. Psychiatry*, 48, 210-221.
7. Munro et al. Submitted.
8. Blair et al. (2004). *Pers. Individ. Differ.*, 37, 1179-1182.

Acknowledgements: Funded by the Natural Sciences and Engineering Research Council of Canada. Thanks to James Desjardins, Leslie McGregor, and Matt Shane for their assistance and to participants and staff at the Oakridge Division of the Mental Health Centre Penetanguishene