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# Exploring the neurological substrate of emotional and social intelligence

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## Summary

**The somatic marker hypothesis posits that deficits in emotional signalling (somatic states) lead to poor judgment in decision-making, especially in the personal and social realms. Similar to this hypothesis is the concept of emotional intelligence, which has been defined as an array of emotional and social abilities, competencies and skills that enable individuals to cope with daily demands and be more effective in their personal and social life. Patients with lesions to the ventromedial (VM) prefrontal cortex have defective somatic markers and tend to exercise poor judgment in decision-making, which is especially manifested in the disadvantageous choices they typically make in their personal lives and in the ways in which they relate with others. Furthermore, lesions to the amygdala or insular cortices, especially on the right side, also compromise somatic state activation and decision-making. This suggests that the VM, amygdala and insular regions are part of a neural system involved in somatic state activation and decision-making. We hypothesized that the severe impairment of these patients in real-life decision-making and an inability to cope effectively with environmental and social demands would be reflected in an abnormal level of emotional and social intelligence. Twelve patients with focal, stable bilateral lesions of the VM cortex or with right unilateral lesions of the amygdala or the right insular cortices, were tested on the Emotional Quotient Inventory (EQ-i), a standardized psychometric measure of various aspects of emotional and social intelligence. We also examined these patients with various other procedures designed to measure decision-making (the Gambling Task), social functioning, as well as personality changes and psychopathology; standardized neuropsychological tests were applied to assess their cognitive intelligence, executive functioning, perception and memory as well. Their results were compared with those of 11 patients with focal, stable lesions in structures outside the neural circuitry thought to mediate somatic state activation and decision-making. Only patients with lesions in the somatic marker circuitry revealed significantly low emotional intelligence and poor judgment in decision-making as well as disturbances in social functioning, in spite of normal levels of cognitive intelligence (IQ) and the absence of psychopathology based on DSM-IV criteria. The findings provide preliminary evidence suggesting that emotional and social intelligence is different from cognitive intelligence. We suggest, moreover, that the neural systems supporting somatic state activation and personal judgment in decision-making may overlap with critical components of a neural circuitry subserving emotional and social intelligence, independent of the neural system supporting cognitive intelligence.**

**Keywords**: social cognition; decision-making; emotion; somatic markers; emotional intelligence

**Abbreviations**: BVRT = Benton Visual Retention Test-Revised; COWA = Controlled Oral Word Association Test; EQ = emotional quotient; EQ-i = Emotional Quotient Inventory; MMPI = Minnesota Multiphasic Personality Inventory; RAVLT = Rey Auditory Verbal Learning Test; TMT = Trail-making Test; VM = ventromedial; WAIS = Wechsler Adult Intelligence Scale; WCST = Wisconsin Card Sorting Test

## Introduction

Patients with lesions to the ventromedial (VM) cortex are subject to impaired personal judgment in decision-making, which is frequently manifested in the manner in which they relate with others. This specific type of impairment is observed even in cases in which cognitive capacity (IQ) falls within the normal or even above-normal range. In spite of normal intellectual capacity, moreover, these patients also

reveal a compromised ability to experience, understand, express and effectively use emotions (Damasio, 1994). In essence, they are cognitively intelligent but typically behave in an unintelligent manner with respect to exercising judgment in making decisions of a personal and interpersonal nature. Based on these clinical observations, the somatic marker hypothesis has been proposed to explain this specific type of impairment (Damasio, 1994). The somatic marker hypothesis outlines a neurological explanation for decision-making impairment. This hypothesis suggests that decision-making is a process that depends on emotional signals, which are defined as bio-regulatory responses aimed at maintaining homeostasis and ensuring survival. The roots of this concept, as well as those of emotional intelligence, can be traced back to Charles Darwin's early research that culminated in the publication of the first scientific work on the topic titled *The expression of the emotions in man and animals* (Darwin, 1872).

The somatic marker hypothesis specifies a number of structures and operations required for the normal function of decision-making. In brief, the hypothesis posits that the amygdala is a critical substrate in the system that triggers somatic states activated by primary inducers (Bechara *et al*., 1999). Primary inducers are unconditioned stimuli that are innately set as pleasurable or aversive, or conditioned stimuli, which when they are present in the immediate environment, automatically and obligatorily elicit a somatic response. Secondary inducers are entities generated by recall or by thought, and they elicit a somatic response when brought to memory (Damasio, 1995). Once somatic states from primary inducers are induced, signals from them are relayed to the brain. Representations of these signals can remain covert at the level of the brainstem, or can reach the parietal cortices (insular/SI, SII) and posterior cingulate cortices and be perceived as a feeling (Maddock, 1999; Damasio *et al*., 2000). When we process a secondary inducer, i.e. recall an event associated with a feeling, we may re-enact the somatic state characteristic of the feeling. The VM cortex is a trigger structure for somatic states from secondary inducers (Bechara *et al*., 1999). Decision-making is a complex process that sometimes involves a conflict between a primary inducer (which may be positive) and a secondary inducer (which may be negative). Sometimes, the conflict could be between only secondary inducers (e.g. a positive thought versus a negative thought). Regardless of how they are triggered, once somatic states induced by primary and/or secondary inducers are enacted in the body, all these somatic states, which can be either positive or negative, are summed into one overall somatic state (figuratively and literally speaking). This overall somatic state then provides signals to the brain that participate in at least two functions. (i) In one function, it provides a substrate for feeling the overall emotional state, possibly via the insular/somatosensory cortices (SI, SII). (ii) In the other function, it provides a substrate for biasing the decision to select a response (Damasio, 1999). This biasing effect may occur covertly at the level of the striatum, in which case the person acts without a conscious decision to do so. The biasing effect may also occur overtly at the level of the lateral orbitofrontal cortex when the person favours a plan of action, and at the anterior cingulate when the person executes a plan of action that is under volitional control.

The concept of the somatic maker hypothesis is thought to overlap with the concept of emotional intelligence regarding the use of emotions to guide human behaviour (Bechara et al., 2000*a*). The conceptural nexus between the somatic marker hypothesis and emotional intelligence can be seen in the way the latter has been defined by some researchers (Bar-On, 2000, 2001): A multifactorial array of interrelated emotional, personal and social competencies that influence our ability to actively and effectively cope with daily demands. Most conceptualizations of this construct address one or more of the following basic components: (i) the ability to be aware of and express emotions; (ii) the ability to be aware of others' feelings and to establish interpersonal relationships; (iii) the ability to manage and regulate emotions; (iv) the ability to realistically and flexibly cope with the immediate situation and solve problems of a personal and interpersonal nature as they arise; and (v) the ability to generate positive affect in order to be sufficiently self-motivated to achieve personal goals. The concept of emotional intelligence is closely related to the concept of social intelligence. This conceptual proximity is evident in the way in which social intelligence was first defined by Thorndike in 1920 (Thorndike, 1920)—the ability to perceive one's own and others' internal states, motives and behaviours, and to act toward them optimally on the basis of that information. Because of the similarity between both concepts, some psychologists have suggested that they may relate to different aspects of the same construct and could actually be referred to as 'emotional and social intelligence' (Bar-On, 2000, 2001). With respect to the present study, moreover, it is important to note that both concepts and closely related constructs are thought by some to be based on a cognitive schemata (Mayer and Salovey, 1997; Taylor *et al*., 1997; Lane, 2000; Zirkel, 2000). This means that what is perceptually scanned in one's immediate environment, together with one's emotional reaction to that which is perceived, may then be processed, evaluated and understood in order to effectively guide interpersonal behaviour. In the somatic marker hypothesis, it is important to note that there is also a covert route that mediates behaviour, which does not require information to be processed, evaluated and understood. However, it is not yet certain if such a covert route applies to the emotional and social intelligence construct as well.

Together with cognitive intelligence, emotional and social intelligence form important components of general intelligence. One of the major differences between the two is that the former is thought to relate primarily to higher order mental processes like reasoning, while the latter focuses more on perceiving, immediate processing and applying emotional and social content, information and knowledge. It has also been suggested that another fundamental difference between

**Table 1** *Demographic data of participating subjects*

| Demographic data | Control group | Experimental group | *Z* | *P*-level (2-tailed) |
|---|---|---|---|---|
| Total number of participants | 11 | 12 | – | – |
| Gender (male/female) | 4/7 | 7/5 | –/– | –/– |
| Age (years) | 47.1 | 43.5 | 0.46 | 0.644 |
| Age range (years) | 24–74 | 21–63 | – | – |
| Education (years) | 14.6 | 13.7 | 0.85 | 0.398 |
| Handedness (right/left) | 10/1 | 12/0 | –/– | –/– |

The Mann–Whitney *U* test was applied to compare the average age and years of education between the control group and the experimental group for significant differences.



the two may be that cognitive intelligence is more cortically strategic in nature, while emotional and social intelligence is more limbically tactical for immediate behaviour suited more for survival and adaptation (Goleman, 1995; Bar-On, 1997*a*; Stein and Book, 2000). However, thus far these theories are supported more by supposition than by empirical findings.

One of the primary purposes of this study was to provide empirical evidence in support of the hypothesis that emotional and social intelligence is different from cognitive intelligence, in that these two major components of general intelligence are supported by separate neural substrates. Furthermore, we hypothesized that the neural systems that support emotional and social intelligence overlap with neural systems subserving somatic state activation and personal judgment in decision-making, which are separate from the neural systems supporting cognitive intelligence. We predicted that patients with lesions in critical components of the somatic marker circuitry (i.e. amygdala, VM prefrontal and insular/somatosensory cortices), who demonstrate severe impairments in real-life decision-making and an inability to cope effectively with environmental and social demands, would also demonstrate an abnormally low level of emotional and social intelligence. Specifically, we predicted that these patients would present significantly lower scores and ratings than the control group (patients with lesions outside the neural circuitry involved with the somatic state activation) on measures of emotional intelligence, decision-making as well as overall social functioning, despite maintaining normal levels of cognitive intelligence.

## Methods

Twenty-three neurological patients were selected and divided into an experimental group and a control group based on the presence or absence of brain injury along the circuitry thought to mediate somatic state activation and decision-making.

Because of the relatively small sample sizes involved in the present study, non-parametric statistics were applied; the Mann–Whitney *U* test was employed to examine the groups being compared for significant differences (Siegal, 1956). In tandem with this conservative approach, a two-tailed evaluation was applied in interpreting the probability levels of the results. It was decided, moreover, to discuss significant differences on Emotional Quotient Inventory (EQ-i) subscale scores (e.g. emotional self-awareness) only if the parent composite scale score (i.e. intrapersonal EQ) proved to be significantly different when comparing the independent groups being examined.

### *Subjects*

The experimental and control groups were matched with respect to gender, age, level of education and handedness. As can be seen in Table 1, the subjects were predominantly right-handed, in their mid-forties and had 14 to 15 years of education with no significant difference between the groups regarding age or education. The experimental group included seven males and five females, and the control group comprised four males and seven females.

Subjects with brain lesions were selected from the Patient Registry of the University of Iowa's Division of Cognitive Neuroscience. All brain-damaged subjects had undergone basic neuropsychological and neuroanatomical characterization according to the standard protocols of the Benton Neuropsychology Laboratory (Tranel, 1996) and the Laboratory of Neuroimaging and Human Neuroanatomy (Damasio and Damasio, 1989; Damasio and Frank, 1992; Damasio, 1995). All subjects provided informed consent, which was approved by the appropriate human subject committees at the University of Iowa. Based on clinical interviews, none of the subjects in this study had a history of mental retardation, psychiatric disorder, substance abuse, learning disability or systemic disease that might affect the CNS.

The selection of subjects with brain lesions conformed to the following criteria: (i) a stable and chronic lesion at least 3 months post onset; and (ii) involvement of a brain region that either included (the experimental group) or excluded (the control group) structures of the somatic marker circuitry as shown below. The experimental, neuropsychological and neuroanatomical studies were all conducted when the subjects were in the chronic phase of recovery (i.e. >3 months post lesion onset). Data collection for the various studies was contemporaneous for each subject (i.e. experimental, neu-

ropsychological and neuroanatomical data for a given subject were collected at the same time or at a very close point in time).

### *The experimental group*

This group included patients with lesions in either the VM prefrontal cortex, the right insular/somatosensory cortex or the right amygdala.

*VM lesions*. Six subjects had lesions involving the VM cortex bilaterally. In four subjects, the damage was caused by a stroke. In the other two subjects, the damage was caused by a frontal lobe meningioma that was surgically resected and removed. In all six subjects, the damage was bilateral and involved the anterior and posterior sectors of the VM cortex. In two of the subjects, the damage extended far posteriorly and most likely included the basal forebrain. In one subject, the lesion extended superiorly above the genu of the corpus callosum in both hemispheres. In three of the subjects, the lesions extended anteriorly and involved the frontopolar region. In two of the subjects, the lesions were asymmetrical in that the damage was more extensive on the right relative to the left. In all subjects, the dorsolateral sectors of the prefrontal cortex were intact.

*Amygdala lesions*. Three subjects had lesions involving the amygdala. Patients with bilateral amygdala damage are extremely rare. Therefore, we decided to select patients with unilateral amygdala damage. Because the somatic marker hypothesis emphasizes the role of the right hemisphere as opposed to the left in somatic state activation and in light of recent support of this view in relation to patients with prefrontal cortex damage (Tranel *et al.*, 2000; Manes *et al.*, 2002), we selected patients with unilateral damage to the right amygdala.

In all three subjects, the aetiology was a right temporal lobectomy in order to treat an intractable seizure disorder. In all three subjects, the amygdala was completely removed. The entorhinal cortex overlaying the amygdaloid nucleus was damaged. However, there was minimal damage to the surrounding anterior sector of the hippocampal formation.

*Insular/somatosensory (SI, SII) lesions*. Three subjects had unilateral lesions involving the right insular/somatosensory (SI, SII) cortices. In all subjects, the aetiology was a right middle cerebral artery stroke. And in all three subjects, the insular cortex was damaged. There was also extensive damage to the superior and inferior parietal lobules, which include the somatosensory (SI, SII) cortices.

In two of the subjects, the damage extended to the right dorsolateral prefrontal cortex and included the right precentral gyrus, but the cortex anterior to it was spared. There was also damage to the superior temporal gyrus. In one subject, the lesion included the right insular cortex and inferior parietal lobule, but did not extend into the prefrontal cortex or the temporal lobe.

### *The control group*

This group included patients with lesions outside the neural circuitry thought to mediate somatic state activation and decision-making. In all 11 subjects, the aetiology of the lesion was a stroke. In four subjects, the damage included any of the following regions: the superior and/or middle frontal gyri in the right prefrontal cortex, the right precentral gyrus, the right paracentral lobule without damage below the body of the corpus callosum or extension to the frontal pole. In two subjects, the lesion involved similar territories in the left hemisphere. In three subjects, the lesions involved the posterior sector of the superior and/or middle temporal gyrus on the right. In one subject, the lesion involved the posterior sector of the middle temporal gyrus on the left. In one subject, the damage involved the right occipital cortex but spared the somatosensory and insular cortices.

## *Measures*

The vagary of subject availability, being neurologically ill, not easily accessible, etc. produced some limitations on data collection but without interfering systemically with the outcome. Every subject completed the EQ-i and the Gambling Task (see below) in the present study. Almost all the subjects in the experimental group completed the social functioning, cognitive intelligence and executive functioning tests. However, a small number of subjects in the control group did not complete all these tests. Quite often, this was because the clinician could not justify administering these specific tests for these particular subjects. This was due to the nature of each subject's lesion, in which there was no suspicion that the subject would have problems in the domains measured by the clinical tests involved. Although not everyone was available to be tested with all of the measures described below, data collection for the critical group, the experimental group, was nearly complete. The missing data from some subjects, who were mostly those in the control group, do not systematically affect the outcome of the results because nonparametric statistics were applied which are designed specifically for examining small samples (Siegal, 1956).

### *Emotional intelligence*

The Bar-On EQ-i was used to assess emotional intelligence (Bar-On, 1997*a*). The EQ-i is a self-report measure of emotionally and socially intelligent behaviour, which provides an estimate of one's underlying emotional and social intelligence. A more detailed discussion of the psychometric properties of this instrument and how it was developed is found in the EQ-i technical manual (Bar-On, 1997*b*) and elsewhere (Plake and Impara, 1999). In brief, the EQ-i

comprises 133 items and employs a five-point Likert scale with a textual response format ranging from 'very seldom or not true of me' to 'very often true of me or true of me'. A list of the inventory's items is included in the EQ-i technical manual (Bar-On, 1997*b*). The subject's responses render a total emotional quotient (EQ) score and the following five composite scale scores give 15 subscale scores in all:

(i) Intrapersonal EQ (comprising self-regard, emotional self-awareness, assertiveness, independence and self-actualization);

(ii) Interpersonal EQ (comprising empathy, social responsibility and interpersonal relationship);

(iii) Stress management EQ (comprising stress tolerance and impulse control);

(iv) Adaptability EQ (comprising reality-testing, flexibility and problem-solving); and

(v) General mood EQ (comprising optimism and happiness).

A brief description of the emotional and social intelligence competencies measured by the 15 subscales is given in the Appendix. The EQ-i has a built-in correction factor which automatically adjusts the scale scores based on scores obtained from its two validity indices (the positive impression and negative impression scales); this is an important psychometric factor for self-report measures in that it reduces the distorting effects of response bias, thereby, increasing the accuracy of the results obtained. Also, this correction factor is of particular importance in the current application of the EQ-i because some of the brain-damaged subjects' self-awareness of their acquired deficits is limited (i.e. anosognosia). Raw scores are computer-tabulated and automatically converted into standard scores based on a mean of 100 and standard deviations of 15. It is important to stress that the EQ-i is acknowledged as a valid measure of emotional intelligence based on independent review (Plake and Impara, 1999). Moreover, the EQ-i is significantly correlated with other measures that tap various aspects of this construct, for example with the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT) (0.46), Trait Meta-Mood Scale (TMMS) (0.58), Emotional Intelligence Questionnaire (EIQ) (0.63) and the 20-item Toronto Alexithymia Scale (TAS-20) (–0.72) (Bar-On, 2000). This means that the EQ-i is tapping—relatively well—what these other measures are tapping (i.e. various aspects of emotional intelligence).

All participants completed the EQ-i ($n$ = 23).

### *Personal judgment in decision-making*

The ability to exercise personal judgment in decision-making was assessed by the Gambling Task. A detailed account of this measure is given elsewhere (Bechara *et al*., 1994, 2000*b*). In brief, subjects are required to select a total of 100 cards from four packs labelled A, B, C and D. The subject's decision to select from one pack versus another is largely influenced by various schedules of immediate reward and future punishment. These schedules are pre-programmed and known only to the examiner and entail a number of basic game rules that must be adhered to by the subject. First, every time the subject selects a card from pack A or B, the subject receives $100; and every time the subject selects a card from pack C or D, the subject receives $50. However, in each of the four packs, subjects encounter unpredictable punishments (money loss). The punishment is more severe in the high-paying packs A and B, and less severe in the low-paying packs C and D. Overall, selections from packs A and B are disadvantageous because they cost more in punishments in the long run, i.e. one loses $250 every 10 cards. Packs C and D are advantageous because they result in an overall gain in the long run, i.e. one wins $250 every 10 cards.

All participants completed the Gambling Task ($n$ = 23).

### *Social functioning*

Post-morbid employment status, social functioning, interpersonal relationships and social standing were evaluated with a series of semi-structured interviews and rating scales described in detail by Tranel and colleagues (Tranel *et al*., 2002). Briefly, these entailed a comprehensive assessment by a clinical neuropsychologist (who was unaware of the objectives and design of the current study) of each patient's post-morbid employment status, social functioning, interpersonal relationships and social standing. For each of these domains, the extent of social change or impairment for each patient was rated on a three-point scale on which one corresponded to 'no change or impairment'; two corresponded to 'moderate change or impairment'; and three corresponded to 'severe change or impairment.' For each patient, a Total Social Change score was then calculated by summing the four scores from each of the domains. Higher overall scores are indicative of greater levels of change (impairment). Sixteen participants (10 experimental and six control subjects) were assessed for post-morbid social functioning.

### *Cognitive intelligence, perception, memory and executive functioning*

Subjects were assessed according to standard protocols of the Benton Neuropsychology Laboratory (Tranel, 1996). This included standardized measurement of cognitive intelligence, perception, memory and executive functioning.

*Cognitive intelligence*. This was measured with the Wechsler Adult Intelligence Scale (WAIS-III); 21 subjects (12 experimental and nine control) were administered the WAIS.

*Perception*. This was measured with the Benton Facial Discrimination Test and the Benton Judgment of Line Orientation Test; 18 subjects (10 experimental and eight control) completed these tests.

**Table 2** *Neuropsychological test scores for the control and experimental groups*

| Neuropsychological tests | Control group ($n$ = 11) | Experimental group ($n$ = 12) | $Z$ | $P$-level (2-tailed) |
|---|---|---|---|---|
| Cognitive intelligence | | | | |
| WAIS-III: | | | | |
| Full IQ | 97.7 | 105.3 | 1.17 | 0.241 |
| Verbal IQ | 99.2 | 107.9 | 1.32 | 0.186 |
| Performance IQ | 95.7 | 102.8 | 1.42 | 0.155 |
| Perception | | | | |
| Benton faces | 44.4 | 43.9 | 0.50 | 0.620 |
| Benton lines | 25.4 | 24.6 | 0.81 | 0.420 |
| Memory | | | | |
| WAIS: | | | | |
| Digit span | 11.0 | 10.8 | 0.16 | 0.869 |
| BVRT: | | | | |
| Correct | 7.4 | 7.4 | 0.12 | 0.908 |
| Errors | 3.8 | 4.0 | 0.58 | 0.565 |
| RAVLT: | | | | |
| Trial 1 to 5 | 10.1 | 12.0 | 1.53 | 0.127 |
| 30 minute recall | 8.1 | 9.3 | 0.46 | 0.648 |
| Recognition | 28.9 | 28.8 | 0.91 | 0.361 |
| Complex figure (Rey-O): | | | | |
| Copy | 32.2 | 30.9 | 0.55 | 0.585 |
| Delay | 20.7 | 19.5 | 0.22 | 0.827 |
| Executive functioning | | | | |
| Trails-making test A | 34.8 | 39.6 | 0.06 | 0.957 |
| Trails-making test B | 86.8 | 79.9 | 0.33 | 0.745 |
| WCST: | | | | |
| Perseverative errors | 10.2 | 17.7 | 0.44 | 0.662 |
| Categories | 5.8 | 5.1 | 0.71 | 0.476 |
| COWA | 37.0 | 40.4 | 0.53 | 0.596 |
| Personality/psychopathology | | | | |
| MMPI: | | | | |
| Scale 1 (Hs) | 57.3 | 57.6 | 0.34 | 0.732 |
| Scale 2 (D) | 53.0 | 63.0 | 1.26 | 0.209 |
| Scale 3 (Hy) | 64.3 | 57.6 | 0.69 | 0.493 |
| Scale 4 (Pd) | 58.7 | 61.9 | 1.03 | 0.304 |
| Scale 5 (Mf) | 52.7 | 55.0 | 0.57 | 0.568 |
| Scale 6 (Pa) | 50.3 | 57.1 | 0.92 | 0.359 |
| Scale 7 (Pt) | 49.3 | 61.3 | 1.38 | 0.168 |
| Scale 8 (Sc) | 52.0 | 63.3 | 1.48 | 0.139 |
| Scale 9 (Ma) | 54.3 | 53.9 | 0.23 | 0.818 |
| Scale 0 (Si) | 49.7 | 52.9 | 0.34 | 0.731 |

The Mann–Whitney $U$ test was applied to compare the scores for significant differences between the groups.

*Memory*. This was measured with the Rey Auditory Verbal Learning Test (RAVLT), the Benton Visual Retention Test-Revised (BVRT), and the Complex Figure Test; 20 subjects (10 experimental and 10 control) were assessed with these instruments.

*Executive functioning*. This was measured with the Wisconsin Card Sorting Test (WCST), the Trail-making Test (TMT) and the Controlled Oral Word Association Test (COWA); 16 subjects (10 experimental and six control) completed the WCST and the TMT, and 20 (10 experimental and 10 control) were tested with the COWA.

### *Personality and psychopathology*

Personality and psychopathology was assessed with the Minnesota Multiphasic Personality Inventory (MMPI) (Butcher *et al*., 1989). Ten subjects (seven experimental and three control) completed the MMPI.

## Results

### *Differences in cognitive intelligence between the control and experimental groups*

There are no significant differences between the control and experimental groups with respect to cognitive intelligence,

**Table 3** *Differences in post-morbid social functioning between the control and experimental groups*

| Post-morbid changes in: | Control group ($n$ = 6) | Experimental group ($n$ = 10) | $Z$ | $P$-level (2-tailed) |
|---|---|---|---|---|
| Employment | 1.17 | 2.70 | –3.11 | 0.002 |
| Social functioning | 1.00 | 2.30 | –2.84 | 0.005 |
| Interpersonal relationships | 1.00 | 2.40 | –2.87 | 0.004 |
| Social status | 1.00 | 1.90 | –3.40 | 0.001 |
| Total social change | 4.17 | 9.30 | –3.00 | 0.003 |

The Mann–Whitney $U$ test was applied to compare scores for significant differences.

**Table 4** *Differences in emotional intelligence between the control and experimental groups*

| EQ-i scales (emotional intelligence) | Control group ($n$ = 11) | Experimental group ($n$ = 12) | Z | $P$-level (2-tailed) |
|---|---|---|---|---|
| Total EQ | 101.1 | 82.3 | 3.33 | 0.001 |
| Intrapersonal EQ | 100.0 | 81.8 | 3.23 | 0.001 |
| Self-regard | 99.1 | 83.8 | 2.40 | 0.016 |
| Emotional self-awareness | 100.9 | 90.1 | 1.48 | 0.139 |
| Assertiveness | 103.6 | 82.6 | 3.21 | 0.001 |
| Independence | 97.7 | 87.3 | 1.58 | 0.115 |
| Self-actualization | 99.4 | 86.8 | 2.25 | 0.024 |
| Interpersonal EQ | 99.6 | 91.6 | 1.36 | 0.175 |
| Empathy | 98.6 | 89.8 | 1.24 | 0.216 |
| Social responsibility | 101.5 | 95.3 | 1.14 | 0.254 |
| Interpersonal relationship | 98.8 | 92.8 | 0.83 | 0.406 |
| Stress management EQ | 104.8 | 89.1 | 2.62 | 0.009 |
| Stress tolerance | 100.1 | 83.2 | 2.56 | 0.011 |
| Impulse control | 108.3 | 96.9 | 2.13 | 0.033 |
| Adaptability EQ | 100.0 | 86.3 | 2.28 | 0.023 |
| Reality testing | 99.8 | 91.0 | 1.08 | 0.280 |
| Flexibility | 100.3 | 86.8 | 2.38 | 0.017 |
| Problem-solving | 100.6 | 88.3 | 2.16 | 0.031 |
| General mood EQ | 99.9 | 83.3 | 3.27 | 0.001 |
| Optimism | 99.0 | 83.5 | 3.02 | 0.003 |
| Happiness | 100.9 | 85.8 | 2.71 | 0.007 |

Emotional intelligence was assessed by EQ-i and the Mann–Whitney $U$ test was applied to compare scores for significant differences.

executive functioning, perception, memory or signs of psychopathology (see Table 2). In addition to a lack of significant difference between the control group and the experimental group regarding the level of their cognitive intelligence as can be seen in Table 2 ($Z$ = 1.17, $P$ = 0.241), it is also important to point out that no significant correlation was found between cognitive intelligence and emotional intelligence ($r$ = 0.08, $P$ = 0.740) for the clinical sample examined in the present study.

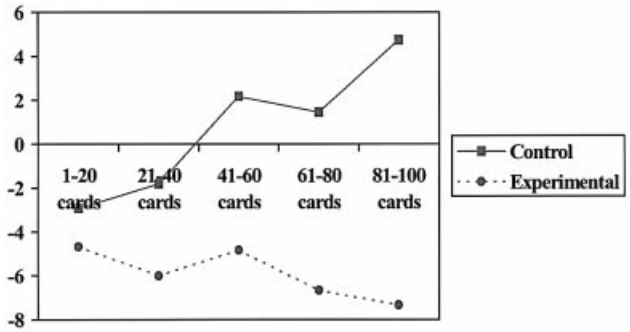


**Fig. 1.** The ratio of advantageous (+) to disadvantageous (–) decisions made in the Gambling Task by the control group ($n$ = 11) and the experimental group ($n$ = 12) progressing from the first 20 to the last 20 cards selected.

## *Differences in decision-making, social functioning and emotional intelligence between the control and experimental groups*

A comparison of advantageous (+) to disadvantageous (–) choices made in the Gambling Task for the first 40 cards selected did not reveal significant differences between the experimental and control groups (–10.67 versus –04.73, respectively; $Z$ = 1.52, $P$ = 0.128). During the next and final 60 selections, however, significant differences began to appear between the two groups with the experimental group making more disadvantageous than advantageous choices; this ratio was reversed for the control group who began to make more advantageous than disadvantageous choices (–18.83 versus +08.36, respectively; $Z$ = 3.67, $P$ < 0.000). This increase in exercising better judgment in decision-making among subjects in the control group appears to follow a typical learning curve that would be expected; however, it is

important to note that this normal learning process did not occur among the subjects in the experimental group whose personal judgment tends to get worse as can be seen in Fig. 1. In addition to indicating the existence of a neurological substrate mediating the somatic marker hypothesis, this suggests that it takes ~40 attempts of trial and error before people, who are not damaged along the targeted somatic marker circuitry, to catch on to the rules of the game and to do what is more advantageous for them. Based on these findings and in an attempt to obtain the most accurate evaluation of differences between the groups that were examined, it was decided to focus only on the last 60 attempts in the Gambling Task when comparing personal judgment in decision-making with emotional intelligence and overall social functioning in the present study.

Post-morbid social functioning was also found to be significantly worse for the experimental group compared with the control group (see Table 3).

Table 4 reveals that subjects in the experimental group have significantly lower emotional intelligence than those in the control group (82.3 versus 101.1, respectively; $Z = -3.33$, $P = 0.001$). A review of these results suggest that the key emotional intelligence competencies involved appear to be the ability to be aware of oneself (self-regard), to express oneself (assertiveness), to manage and control emotions (stress tolerance and impulse control), to adapt flexibly to change (flexibility) and to solve problems of a personal nature as they arise (problem-solving), as well as the ability to motivate oneself and mobilize positive affect (self-actualization, optimism and happiness).

A precursory examination of the differences between the control group and all of the three subgroups forming the experimental group reveals significant differences with respect to emotional and social intelligence (those with damage to the VM prefrontal lobe (101 versus 92; $Z = 2.22$, $P = 0.026$), the right amygdala (101 versus 80; $Z = 2.43$, $P = 0.015$) and, especially, to the right insular/somatosensory cortices (101 versus 65; $Z=2.58$, $P = 0.010$)). However, a closer examination of the specific differences between these subgroups is not justified because of the very small sample sizes involved.

## Discussion

Only those subjects with injury to the somatic marker circuitry (the experimental group) revealed significantly low emotional intelligence and poor judgment in decision-making as well as disturbances in social functioning, in spite of normal levels of cognitive intelligence (IQ) and in the absence of psychopathology. Specifically, the experimental group made significantly more disadvantageous decisions (on the Gambling Task) than the control group and their personal judgment got worse rather than better as time went on, i.e. they failed to make advantageous choices and to learn from experience. In addition to obtaining significantly lower scores on the Gambling Task, the experimental group demonstrated many disturbances in social functioning, and they received significantly lower scores on the EQ-i indicating impaired emotional and social intelligence. Yet, there were no differences between the experimental and control groups with respect to cognitive capacity (IQ), executive functioning, perception, memory or signs of psychopathology. Similarly, there were no differences between the two groups with respect to demographic factors. Thus, the differences between the experimental and control groups with respect to emotional and social intelligence (EQ) exist in spite of the fact that the two groups were well matched on demographic and cognitive grounds. It was also demonstrated that there was no significant correlation between IQ and EQ in the clinical sample studied in the present study. In addition to reconfirming the neurological substrate that mediates somatic state activation and decision-making, these findings support our hypothesis that emotional and social intelligence is fundamentally different from cognitive intelligence. Moreover, the neural systems that support emotional and social intelligence appear to overlap with the neural systems subserving somatic state activation and personal judgment in decision-making, which are apparently separate from the neural systems supporting cognitive intelligence.

The key emotional intelligence competencies affected by damage to the neural circuitry of somatic markers appear to be the ability to be aware of oneself and one's emotions, to express oneself and one's feelings, to manage and control emotions, to adapt flexibly to change and solve problems of a personal nature, as well as the ability to motivate oneself and mobilize positive affect. Indeed, self-regard (accurate self-awareness) and, especially assertiveness (self-expression), stand out as those competencies that are affected most by brain injury to the neural circuitry being examined in the present study. This is understandable in that these are two of the most important emotional intelligence competencies.

It is important to note that the specific type of brain injury sustained by subjects belonging to the experimental group usually produces a certain degree of anosognosia (i.e. lack of self-awareness of acquired impairments). Given the reliance of the EQ-i on self-report, the question arises as to whether this instrument can provide a valid measure of emotional and social intelligence when used with this particular group of clinical subjects. The issue of self-awareness of acquired impairments is critical in situations where self-report measures fail to detect acquired impairments in tested individuals. This situation often occurs in the case of VM patients as was previously noted, in which case collateral information is usually needed in order to document changes in the personality and social behaviour of impaired patients (Barrash *et al*., 2000). However, in the case of the EQ-i, the instrument proved to be successful in detecting abnormal levels of emotional and social intelligence in the target subjects. This suggests that the instrument has adequate construct validity when used with individuals whom otherwise may be unaware of the limitations of their own emotional and social abilities. Indeed, low scores—particularly on the two scales of self-

regard (accurate self-awareness) and assertiveness (self-expression)—most likely mean that these subjects possess low self-awareness consistent with their symptomatology; their scores on these two scales would have been even lower without the EQ-i's correction factor that automatically adjusts scale scores to compensate for various types of inaccuracies in responding that occur for one reason or another.

The findings of the present study indicate that poor personal judgment in decision-making is related to deficiencies in emotional intelligence, in spite of average or above average levels of cognitive intelligence. Relative to patients with brain damage in areas outside of the neural circuitry studied in the present study (the control group), those who exercise poor judgment in decision-making linked to impaired somatic state activation (the experimental group) are less emotionally intelligent based on lower EQ-i scores. Furthermore, poor decision-making appears to be related to an inadequate knowledge of who one is (accurate self-awareness), what one wants and how to convey this effectively and constructively (self-expression). Equally important is that subjects who often fail to make the right decision are also less effective in controlling their emotions, in maintaining a positive and optimistic attitude, and in generating and selecting potentially effective solutions.

Emotional and social intelligence provides a valuable approach to understanding why some people behave more intelligently than others, which is often revealed in making the right versus wrong decisions in one's personal life and interactions with others. There are a number of other closely related concepts that are based on the way emotions are perceived, understood and used to guide effective human behaviour like 'emotional awareness' (Lane and Schwartz, 1987), 'empathy' (Brothers, 1990; Preston and de Waal, 2002), 'psychological mindedness' (McCallum, 1989), 'theory of mind' (Gopnik, 1993; Gallup, 1998; Blair, 1999; Frith and Frith, 1999), 'practical intelligence' (Sternberg, 1985), 'successful intelligence' (Sternberg, 1997), etc. Most of these concepts can be considered different components of emotional and social intelligence rather than separate constructs. For example, psychological mindedness is the salutogenic (i.e. non-pathological) end of alexithymia and emotional awareness is a major component of this continuum. While this continuum represents the essence of the intrapersonal aspect of emotional intelligence, empathy and theory of mind represent the essence of social intelligence; both types of intelligence have been combined into one meta construct by some theorists (Gardner, 1983; Goleman, 1995; Bar-On, 2001).

One of the implications of the significant statistical and neurological connections between the somatic marker hypothesis and the concept of emotional intelligence, based on the present findings, is that emotional and social intelligence is a valid construct which is neurally distinct from cognitive intelligence. The major differences between these two important components of intelligence may be that cognitive intelligence is more dependent on cortical structures that support logical reasoning, whereas emotional and social intelligence is more dependent on limbic and related neural systems that support the processing of emotions and feelings. In this study, we have examined clinical subjects who possess average or above average cognitive intelligence and significantly below average emotional intelligence. However, we do not know the impact of below average cognitive intelligence (IQ) on emotional and social intelligence. The neural systems supporting emotional and cognitive intelligence may be completely independent, i.e. impaired cognitive intelligence does not compromise emotional intelligence. Alternatively, the dissociation may only be partial (i.e. impaired emotional intelligence does not compromise cognitive intelligence) as we have shown in this study. However, impaired cognitive intelligence may compromise emotional intelligence. Such asymmetrical relationship between neural systems has been demonstrated before in relation to two functions of the prefrontal cortex: decision-making and working memory. Bachara and colleagues found that VM lesions that impaired decision-making did not compromise working memory, but dorsolateral lesions that impaired working memory did compromise decision-making (Bechara *et al*., 1998). Thus, it would be intriguing to study patients with Down's syndrome or William's syndrome, for example, who have significantly limited cognitive intelligence but are known to be relatively effective in social interactions. Another approach is to examine the level of emotional and social intelligence in neurological patients with impaired cognitive intelligence. In this study, we used the WAIS-III to measure cognitive intelligence, whereas Duncan (2001), for instance, has used more sensitive tasks to measure various aspects of cognitive intelligence, attributing them to the lateral orbitofrontal/dorsolateral prefrontal cortices. Therefore, using these more sensitive measures of cognitive intelligence in future studies will yield additional important information about the relationships between the neural systems that support cognitive versus emotional intelligence.

One of the most important implications of the current findings is that the complex cognitive processes that subserve social competence, which appears to constitute a distinct form of intelligence dedicated to behaviour suited more for survival and adaptation (Goleman, 1995; Bar-On, 1997*b*; Stein and Book, 2000), does not draw upon neural processes specialized for social information. Rather, these processes depend on known brain mechanisms related to emotion and decision-making. Indeed, we have argued that the process of judgment and decision-making depends on systems for: (i) memory, which is supported by high-order association cortices as well as the dorsolateral sector of the prefrontal cortex; (ii) emotion, which is mediated by subcortical limbic structures that trigger the emotional response; and (iii) feelings which are supported by limbic as well as closely associated regions such as the insula, surrounding parietal cortices and the cingulate cortex (Damasio, 1994, 1995, 1999; Bechara *et al*., 2000*a*). Therefore, damage to the systems that impact emotion, feeling and/or memory usually compromise

the ability to make advantageous decisions (Bechara *et al*., 2000*a*). The VM prefrontal cortex links these systems together; when damaged, there are a number of manifestations that occur including alterations of emotional experience and social functioning (Bechara *et al*., 2000*a*). The findings of the present study suggest that emotional and social intelligence has neural roots, which may be associated with these known basic mechanisms of the brain. Impairment of these mechanisms may manifest itself in low levels of emotional intelligence, which comprises a wide array of emotional and social competencies, which can have an ill effect on one's ability to effectively cope with daily demands. Such impairment may include a decrease in one's ability to: (i) be aware of and express oneself; (ii) function interpersonally; (iii) manage and control emotions; (iv) generate positive affect required in achieving personal goals; and (v) cope flexibly with the immediate situation, make decisions and solve problems of a personal and interpersonal nature.

Finally, the findings that emotional intelligence is significantly related to the ability to exercise personal judgment in decision-making help explain why this concept is highly connected with human performance (Bar-On *et al*., 2003). To perform well and be successful in one's professional and personal life apparently requires the ability to make emotionally and socially intelligent decisions more than just having a high IQ.

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### References

Bar-On R. The Bar-On Emotional Quotient Inventory (EQ-i): a test of emotional intelligence. Toronto (Canada): Multi-Health Systems; 1997a.

Bar-On R. The Bar-On Emotional Quotient Inventory (EQ-i): technical manual. Toronto (Canada): Multi-Health Systems; 1997b.

Bar-On R. Emotional and social intelligence: insights from the Emotional Quotient Inventory. In: Bar-On R, Parker JDA, editors. The handbook of emotional intelligence. San Francisco: Jossey-Bass; 2000. p. 363–88.

Bar-On R. Emotional intelligence and self-actualization. In: Ciarrochi J, Forgas JP, Mayer JD, editors. Emotional intelligence in everyday life: A scientific inquiry. Philadelphia: Psychology Press; 2001. p. 82–97.

Bar-On R, Fund S, Handley R. The impact of emotional intelligence on performance. In: Druskat V, Sala F, Mount G, editors. Emotional intelligence and performance at work. San Francisco: Jossey-Bass. In press 2003.

Barrash J, Tranel D, Anderson SW. Acquired personality disturbances associated with bilateral damage to the ventromedial prefrontal region. Dev Neuropsychol 2000; 18: 355–81.

Bechara A, Damasio AR, Damasio H, Anderson SW. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 1994; 50: 7–15.

Bechara A, Damasio H, Tranel D, Anderson SW. Dissociation of working memory from decision making within the human prefrontal cortex. J Neurosci 1998; 18: 428–37.

Bechara A, Damasio H, Damasio AR, Lee GP. Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. J Neurosci 1999; 19: 5473–81.

Bechara A, Tranel D, Damasio AR. Poor judgment in spite of high intellect: neurological evidence for emotional intelligence. In: Bar-On R, Parker JDA, editors. The handbook of emotional intelligence. San Francisco: Jossey-Bass; 2000a. p. 192–214.

Bechara A, Tranel D, Damasio H. Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. Brain 2000b; 123: 2189–202.

Blair RJR. Psychophysiological responsiveness to the distress of others in children with autism. Personality Individ Diff 1999; 26: 477–85.

Brothers L. The neural basis of primate social communication. Motivation Emotion 1990; 14: 81–91.

Butcher JN, Dahlstrom WG, Graham JR, Tellegen A, Kaemmer B. Minnesota Multiphasic Personality Inventory-2 (MMPI-2) manual for administration and scoring. Minneapolis (MN): University of Minnesota Press; 1989.

Damasio AR. Descartes' error: emotion, reason, and the human brain. New York: G. P. Putnam; 1994.

Damasio AR. Toward a neurobiology of emotion and feeling: operational concepts and hypotheses. Neuroscientist 1995; 1: 19–25.

Damasio AR. The feeling of what happens: body and emotion in the making of consciousness. New York: Harcourt Brace; 1999.

Damasio H, Damasio AR. Lesion analysis in neuropsychology. New York: Oxford University Press; 1989.

Damasio H, Frank R. Three-dimensional in vivo mapping of brain lesions in humans. Arch Neurol 1992; 49: 137–43.

Damasio AR, Grabowski TG, Bechara A, Damasio H, Ponto LLB, Parvizi J, et al. Subcortical and cortical brain activity during the feeling of self-generated emotions. Nat Neurosci 2000; 3: 1049–56.

Darwin C. The expression of the emotions in man and animals. London: Murray; 1872.

Duncan J. An adaptive coding model of neural function in prefrontal cortex. Nat Rev Neurosci 2001; 2: 820–9.

Frith CD, Frith U. Interacting minds: a biological basis. Science 1999; 286: 1692–5.

Gallup GGJ. Self-awareness and the evolution of social intelligence. Behav Process 1998; 42: 239–47.

Gardner H. Frames of mind. New York: Basic Books; 1983.

Goleman D. Emotional intelligence. New York: Bantam Books; 1995.

Gopnik A. How we know our minds: the illusion of first-person knowledge of intentionality. Behav Brain Sci 1993; 16: 1–14, 29–113.

Lane RD. Levels of emotional awareness: Neurological, psychological, and social perspectives. In: Bar-On R, Parker JDA, editors. The handbook of emotional intelligence. San Francisco: Jossey-Bass; 2000. p. 171–91.

Lane RD, Schwartz GE. Levels of emotional awareness: a cognitive developmental theory and its application to psychopathology. Am J Psychiatry 1987; 144: 133–43.

Maddock RJ. The retrosplenial cortex and emotion: new insights from functional neuroimaging of the human brain. Trends Neurosci 1999; 22: 310–6.

Manes F, Sahakian B, Clark L, Rogers R, Antoun N, Aitken M, et al. Decision-making processes following damage to the prefrontal cortex. Brain 2002; 125: 624–39.

Mayer JD, Salovey P. What is emotional intelligence: In: Salovey P, Sluyter DJ, editors. Emotional development and emotional intelligence: educational implications. New York: Basic Books; 1997. p. 3–34.

McCallum M. A controlled study of effectiveness and patient suitability for short-term group psychotherapy (doctoral dissertation). Montreal (Canada): McGill University; 1989.

Plake BS, Impara JC, editors. The BarOn Emotional Quotient Inventory (EQ-i). Supplement to the 13th mental measurement yearbook. Lincoln (NE): Buros Institute for Mental Measurement; 1999.

Preston S, de Waal F. Empathy: Its ultimate and proximate bases. Behav Brain Sci 2002; 25: 1–71.

Siegal S. Nonparametric statistics for the behavioral sciences. New York: McGraw-Hill; 1956.

Stein SJ, Book HE. The EQ edge: emotional intelligence and your success. Toronto (Canada): Stoddart Publishing; 2000.

Sternberg RJ. Beyond IQ: a triarchic theory of human intelligence. New York: Cambridge University Press; 1985.

Sternberg RJ. Successful intelligence. New York: Plume; 1997.

Taylor GJ, Bagby RM, Parker JDA. Disorders of affect regulation: alexithymia in medical and psychiatric illness. Cambridge: Cambridge University Press; 1997.

Thorndike EL. Intelligence and its uses. Harper's Magazine 1920; 140: 227–35.

Tranel D. The Iowa-Benton school of neuropsychological assessment. In: Grant I, Adams KM, editors. Neuropsychological assessment of neuropsychiatric disorders, Vol. 1. 2nd ed. New York: Oxford University Press; 1996. p. 81–101.

Tranel D, Bechara A, Damasio H, Damasio AR. Decision-making in patients with unilateral ventromedial prefrontal cortex lesions (abstract). Soc Neurosci Abstr 2000; 26: 549.

Tranel D, Bechara A, Denburg N L. Asymmetric functional roles of right and left ventromedial prefrontal cortices in social conduct, decision-making, and emotional processing. Cortex 2002; 38: 589–612.

Zirkel S. Social intelligence: the development and maintenance of purposive behavior. In: Bar-On R, Parker JDA, editors. The handbook of emotional intelligence. San Francisco: Jossey-Bass; 2000. p. 3–27

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**Appendix I** *The EQ-i scales and what they assess*

| **EQ-i scales** | **The EI competency assessed by each scale** |
|---|---|
| Intrapersonal | |
| Self-regard | To accurately perceive, understand and accept oneself. |
| Emotional self-awareness | To be aware of and understand one's emotions. |
| Assertiveness | To effectively and constructively express one's emotions and oneself. |
| Independence | To be self-reliant and free of emotional dependency on others. |
| Self-actualization | To strive to achieve personal goals and actualize one's potential. |
| Interpersonal | |
| Empathy | To be aware of and understand how others feel. |
| Social responsibility | To identify with one's social group and cooperate with others. |
| Interpersonal relationship | To establish mutually satisfying relationships and relate well with others. |
| Stress management | |
| Stress tolerance | To effectively and constructively manage emotions. |
| Impulse control | To effectively and constructively control emotions. |
| Adaptability | |
| Reality-testing | To objectively validate one's feelings and thinking with external reality. |
| Flexibility | To adapt and adjust one's feelings and thinking to new situations. |
| Problem-solving | To effectively solve problems of a personal and interpersonal nature. |
| General mood | |
| Optimism | To be positive and look at the brighter side of life. |
| Happiness | To feel content with oneself, others and life in general. |