Associations between platelet monoamine oxidase-B activity and acquired colour vision loss in a fish-eating population

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Abstract

Platelet monoamine oxidase-B (MAO-B) has been considered a surrogate biochemical marker of neurotoxicity, as it may reflect changes in the monoaminergic system in the brain. Colour vision discrimination, in part a dopamine dependent process, has been used to identify early neurological effects of some environmental and industrial neurotoxicants. The objective of this cross-sectional study was to explore the relationship between platelet MAO-B activity and acquired colour discrimination capacity in fish-consumers from the St. Lawrence River region of Canada. Assessment of acquired dyschromatopsia was determined using the Lanthony D-15 desaturated panel test. Participants classified with dyschromatopsia (n=81) had significantly lower MAO-B activity when compared to those with normal colour vision (n=32) (26.5±9.6 versus 31.0±9.9 nmol/min/20 μg, P=0.030)). Similarly, Bowman’s Colour Confusion Index (CCI) was inversely correlated with MAO-B activity when the vision test was performed with the worst eye only (r=−0.245, P=0.009), the best eye only (r=−0.188, P=0.048) and with both eyes together (r=−0.309, P=0.001). Associations remained significant after adjustment for age and gender when both eyes (P=0.003) and the worst eye (P=0.045) were tested. Adjustment for heavy smoking weakened the association between MAO-B and CCI in the worst eye (P=0.140), but did not alter this association for both eyes (P=0.006). Adjustment for blood-mercury concentrations did not change the association. This study suggests a relationship between reduced MAO-B activity and acquired colour vision loss and both are associated with tobacco smoking. Therefore, results show that platelet MAO-B may be used as a surrogate biochemical marker of acquired colour vision loss.

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1. Introduction

Monoamine oxidase (MAO, EC 1.4.3.4) is a flavo-enzyme bound to the outer membrane of mitochondria and is involved in the oxidative inactivation of monoamine neurotransmitters including dopamine, serotonin and noradrenaline [49]. There are two isoenzymes, MAO-A and MAO-B, which differ in substrate and inhibitor specificity [32,49]. Both isoenzymes are distributed throughout the central nervous system (CNS) as well as non-neuronal peripheral tissue [54].

The measurement of MAO-B activity in non-neuronal blood platelets has been proposed as a marker for aberrations of the monoaminergic nervous system [45]. Both genetic and environmental factors are known to cause variations in MAO activity levels, which have been linked with personality disorders and some neurological diseases [20,45]. More recently, platelet MAO-B has been investigated for its use as a biochemical marker of neurotoxicity for environmental and occupational contaminants [19,38,52]. Specifically, exposures
to styrene [16], manganese [50], cigarette smoke [3], alcohol [18] and mercury (Hg) [52] have all been associated with reduced platelet MAO-B activity levels in humans. These functional modifications in the surrogate platelet tissue are thought to reflect similar MAO activity changes in the CNS [24,25,38]. However, it remains unclear how an observed reduction in platelet MAO-B activity may be associated with specific sub-clinical neurofunctional variations.

Colour vision discrimination has been used to detect the early neurofunctional alterations following exposure to some neurotoxic chemicals [28]. The retina is particularly sensitive to functional damage by toxicants due to the fenestrated nature of the choriocapillaris [26]. Several neurotoxic compounds including Hg, toluene, styrene, alcohol and tobacco smoke are linked with acquired dyschromatopsia in humans [23,28,40]. This acquired loss of colour discrimination is primarily associated with an impairment of the blue-yellow colour axis, and less frequently with a concomitant loss in the blue-yellow and red-green axis [30]. The Koellner’s rule suggests that colour vision loss in the blue-yellow range is associated with impairments in retinal function (cone neural system) and loss in the red-green range is associated with optic nerve damage [30]. While specific mechanisms of chemical-induced acquired colour vision loss are relatively unknown, it has been suggested that disruption of neurochemical signaling pathways in the retinal cells may be involved [28].

Dopamine is synthesized in the retinal amacrine cells, which is thought to act on both inner and outer retinal neurons [21,41]. It has been established that the dopaminergic system is important for color vision pathways in both laboratory animals and in humans [22,41,47]. Parkinson’s disease, a degenerative disorder affecting dopaminergic neurons in the CNS, disrupts colour vision as well as other visual functions [9,46]. Colour vision impairment can be partially restored following the administration of L-3,4-dihydroxyphenylalanine (L-DOPA), a dopamine precursor and common treatment of Parkinson’s disease [10]. Dopamine in the retina is metabolized and regulated by MAO [44]. While only observed in case studies, overdose and prolonged use of MAO-inhibitor drugs have been linked with retinal and optical neuropathies in humans [1,29,31]. Therefore, adequate MAO function may be important for colour vision and its signaling pathways.

We have previously reported that low-level Hg exposure and heavy smoking is associated with a reduction in platelet MAO-B activity in a fishing community in southern Quebec, Canada [52]. While these associations indicated a possible biochemical effect, it is uncertain whether these changes could predict early neurofunctional effects or neurotoxicity. Because both heavy smoking and Hg exposure have been associated with acquired colour vision loss in previous studies [23,35], our objective was to explore the relationship between platelet MAO-B activity and colour vision performance in the same fishing community. Understanding the interactions between the neurotoxicant, biochemical markers and neurofunctional effects may assist in the (1) identification of mechanisms of early toxic effects and (2) development of novel biomarkers that can be used for risk assessment strategies.

2. Methods

2.1. Population and sampling

This study was conducted as part of the Collaborative Mercury Research Network (COMERN) investigation into Hg exposure and effects on fish-eating communities in Canada. The study population lived in the Lake St. Pierre region in the municipality of Sorel-Tracy (Quebec, Canada). Participants were recruited through the Lake St. Pierre Fisher’s Association, radio stations and local newspapers. This cross-sectional study was carried out from February to April 2003. Persons were excluded from participating if they did not report eating fish from the Lake St. Pierre. Also, due to the known effect of age on colour vision, participants were excluded from analysis if they were over 65 years of age. In total, data from 116 subjects were used. Informed consent was obtained by the participants and coded questionnaires were completed to determine socio-demographic and lifestyle information. Age, gender, smoking habits and vision impairments were collected by questionnaire.

Ethical approval to conduct this study was granted by the McGill University and the University of Quebec at Montreal Ethical Review committees.

2.2. Blood-Hg measurement

Whole blood samples (7 ml) were drawn into heparinized tubes, and analysed for total Hg using cold vapour atomic absorption spectrometry. The detection limit was 0.2 μg/l for blood-Hg analysis. Analysis was carried out at the Centre de Toxicologie du Quebec. Certified reference material was analyzed for quality control purposes.

2.3. Platelet MAO-B measurement

Blood samples were drawn into vacutainer tubes containing EDTA-K2. Blood was centrifuged at 200×g for 10 min to obtain platelet-rich plasma (PRP). The PRP was centrifuged at 3000×g for 25 min at 4°C to pellet platelets. Platelets were washed twice and suspended in phosphate buffer. The concentration of protein in platelets preparations was determined by the Bradford method [7] using bovine serum albumin as the standard. Platelets were stored at −80°C until biochemical assays were performed. Storage conditions and stability of the samples have been tested and the conditions were optimized [51].

MAO-B activity analysis was based on the method described by Krajić [33]. Platelets (20 μg protein) were mixed in a Triton X-100 (0.5%) solution, and then diluted in 3 ml of phosphate buffer saline (50 mM, pH 7.8). Reaction was initiated by the addition of a concentration range of the MAO-B substrate, kynauramine dihydrobromide (1.5–50 μM), followed by a 30 min incubation at 37 °C. Following the incubation, the reaction was stopped by the addition of 5 M perchloric acid and centrifuged at 2000×g for 10 min. The supernatant was diluted with 1 M NaOH and the fluorescence was read at 318 nm excitation and 380 nm emission on a spectrofluorometer. Product concentration was calculated based on the standard
curve of 4-hydroxyquinoline (0–30 nM) and results were expressed in nanomoles of product formed per minute per 20 μg of platelet protein.

2.4. Colour discrimination assessment

Lanthony D-15 desaturated colour panel was used to assess colour discrimination and dyschromatopsia [34]. Participants were asked to place 15 caps in order of chromatic similarity starting from a fixed reference cap. The test was performed first with both eyes together and then with the right and left eye separately. Colour vision loss was classified within types of acquired dyschromatopsia based on Verriest’s classification: type III, a loss in the blue-yellow range; type II, a concomitant loss in the blue-yellow range and red-green range; and type I, a loss in the red-green range [56]. The response patterns were indicative of either acquired or congenital colour vision loss. Participants were classified as dyschromatopic if they had one of the three types of colour vision dysfunction in either the left or right eye.

The Colour Confusion Index (CCI) was calculated and used to estimate acquired colour loss as a score on a continuous scale [6]. A perfect test was given a score of 1.0, while scores greater than 1.0 reflect increasingly incorrect placed caps. The scores from the left eye, right eye and both eyes were determined. The colour vision data were separated into the lowest CCI value representing the best score (best eye), and the highest CCI representing the worst score (worst eye) [11]. Data from obtained from both eyes were also used in the analysis.

2.5. Statistical analysis

Data from MAO-B kinetic analyses were curve fitted using GraphPad Prism (Version 3.02, GraphPad Software Inc., San Diego, CA, USA) to calculate maximum MAO-B velocity (Vmax). Statistical analysis was conducted using SPSS statistical software, Version 10.0, standard version (SPSS Inc., Chicago, IL, USA). Continuous data was analyzed for normality using the Kolmogorov–Smirnov test.

Individuals classified as being congenitally colour blind were excluded from all analyses. Differences between platelet MAO-B activity in the dyschromatopic and normal colour vision groups were determined using a Student’s t-test. Differences among mean MAO-B activity in the normal and specific dyschromatopic category were determined using a one-way analysis of variance (ANOVA). Simple Pearson correlations were determined to evaluate associations between CCI values (best, worst and both eye(s)) and platelet MAO-B activity in both women, men and in the total population. Student’s t-tests were used to detect difference in CCI values between possible confounding factors; gender, heavy smokers (y/n), blood-Hg (above/below 3.4 μg/l). Simple correlations were used to evaluate associations between CCI, age and alcohol consumption.

The participants were separated into tertile groups based on platelet MAO-B activity levels, and mean adjusted CCI scores were compared using General Linear Model (GLM) Univariate procedure. CCI scores (both, worst and best eye(s)) were adjusted for gender and age. Pair-wise comparisons were performed using Bonferroni post-hoc analysis.

Multiple linear regression analyses were also conducted with the CCI values (both, worst and best eye(s)) as the dependent variable and platelet MAO-B activity, gender and age as independent variables. A regression coefficient for MAO-B activity was determined for each eye tested. Both heavy smoking (>14 cigarettes/day) and blood-Hg (>3.4 μg/l) were associated with reduced platelet MAO-B activity in this human population [52]. In order to determine if these environmental variables can explain the association between MAO-B and colour vision, CCI scores were subsequently adjusted for heavy smoking and/or blood-Hg concentrations and regression coefficients for platelet MAO-B activity were determined. Changes in MAO-B regression coefficients and P-values were noted.

3. Results

3.1. Study population description

Three participants were classified as being congenitally colour blind and were excluded from analysis. The study group was composed of 68 men and 45 women, who had an average age (S.D.) of 49 (13) years. Nineteen participants reported smoking more than 14 cigarettes/day and were classified as heavy smokers. The average alcohol intake was 161 g/week and ranged from 0 to 898. Only two participants had reported alcohol intake greater than 500 g/week. Average blood-Hg concentration (S.D.) was 2.5 (3.0) μg/l and 28 participants had levels greater than 3.4 μg/l. MAO-B activity was normally distributed and had a mean (S.D.) of 27.6 (9.8) and a median (range) of 26.6 (5.5–65.4) nmol/min/20 μg.

Four participants reported cataracts and therefore the affected eye was not tested. Of the participants, 35 were considered having normal colour vision, while 81 were dyschromatopic. Of those classified as dyschromatopic, 4 were type I, 24 were type II and 53 were categorized as type III (Table 1). 60% of the women and 79% of the men were classified as dyschromatopic.

3.2. Confounding factors

Age was positively correlated with CCI values in both ($r=0.160$, $P=0.090$), the worst ($r=0.190$, $P=0.046$) and the

<table>
<thead>
<tr>
<th>Colour vision category</th>
<th>Total population</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>n MAO-B</td>
<td>n MAO-B</td>
<td>n MAO-B</td>
<td>n MAO-B</td>
</tr>
<tr>
<td>Normal</td>
<td>32</td>
<td>30.9 (9.9)</td>
<td>14</td>
</tr>
<tr>
<td>Dyschromatopsia</td>
<td>81</td>
<td>26.5 (9.6)*</td>
<td>54</td>
</tr>
<tr>
<td>Type I</td>
<td>4</td>
<td>29.0 (9.5)</td>
<td>4</td>
</tr>
<tr>
<td>Type II</td>
<td>24</td>
<td>26.2 (8.3)</td>
<td>18</td>
</tr>
<tr>
<td>Type III</td>
<td>53</td>
<td>26.5 (10.3)</td>
<td>32</td>
</tr>
</tbody>
</table>

* Indicates significant ($P<0.05$) difference from normal colour vision group as determined using Student’s t-test.
3.3. Association between platelet MAO-B activity and colour vision

Participants classified as dyschromatopic had significantly \((P=0.030)\) lower mean platelet MAO-B activity levels when compared to participants with normal colour vision (Table 1). Although participants with type III (blue/yellow) and type II (combined blue/yellow and red/green) colour vision displayed lower MAO-B activity than those with normal colour vision (Table 1), one-way ANOVA indicated no significant effect \((F(3,112)=1.68, P=0.175)\). MAO-B activity was also significantly lower in dyschromatopic women compared to those with normal colour vision \((P=0.05)\). Although not significant, MAO-B was also lower in males with dyschromatopsia than those with normal colour vision. The three congenitally colour blind participants excluded from analysis had an average platelet MAO-B activity \((SD)\) of 17.9 (5.6) \(\text{nmol/min/20 \mu g}\).

Simple correlations indicated that platelet MAO-B activity was negatively associated with CCI for the best \((P=0.048)\), worst \((P=0.009)\) and both \((P<0.001)\) eye(s) (Table 2). Negative correlations were also observed between MAO-B and CCI when examined separately in men and women, but were significant for both eyes only (Table 2).

Multiple regression analyses were conducted to control for the effects of confounding variables (Table 3). After adjustment for age and gender, lower MAO-B activity remained significantly associated with increased acquired colour vision loss (increased CCI) as determined with the worst and both eyes, but not the best eye. The inclusion of heavy smoking and/or blood-Hg levels did not substantially affect the relationship between MAO-B and CCI score determined when both eyes were used. However, the association between MAO-B and CCI determined from the worst eye was substantially weakened after adjustments were made for the effects of heavy smoking. Adjustment for blood-Hg concentrations did not change the association between MAO-B and colour vision.

In order to further investigate the relationship between MAO-B and colour vision, the population was stratified into tertile groups based on enzyme activity, and the mean CCI (adjusted for age and gender) were compared in each group (Fig. 1). Participants in the lowest MAO-B tertile group had the highest mean CCI for all three eye test conditions, although the difference only reached a significant level when both eyes were tested \((P=0.030)\).

4. Discussion

The major finding from this study is that low platelet MAO-B activity was associated with acquired color vision loss in humans, which can partially be explained by heavy cigarette smoking. This novel finding suggests a continuum of effects relating exposure to a toxicant, to biochemical changes, and to neurofunctional impairment.

To our knowledge, an association between MAO-B and acquired colour vision loss has not been specifically investigated in humans. However, several case reports suggest that use of MAO-inhibitor drugs can result in visual abnormalities and in some cases colour vision impairments [1,29,31]. The mechanism of this toxicity has not been examined, but may

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**Table 2**

<table>
<thead>
<tr>
<th>Eye tested</th>
<th>CCI, mean (S.D.)</th>
<th>Correlations ((r)) with platelet MAO-B activity</th>
<th>Total (113)</th>
<th>Men (68)</th>
<th>Women (45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both eyes</td>
<td>1.26 (0.27)</td>
<td>−0.309***\  −0.244*\  −0.329*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Best eye</td>
<td>1.30 (0.30)</td>
<td>−0.188*\  −0.133\  −0.156</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worst eye</td>
<td>1.48 (0.38)</td>
<td>−0.245***\  −0.173\  −0.244</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\*\(P<0.05\), \**\(P<0.01\), \***\(P<0.001\).

\(a\) CCI for best worst and both eye(s) was log transformed.

---

**Table 3**

<table>
<thead>
<tr>
<th>Colour Confusion Index (a)</th>
<th>Adjustment for confounders (^b, c)</th>
<th>Adjustment for confounders (^b, c) and heavy smoking</th>
<th>Adjustment for confounders (^b, c) and blood-Hg</th>
<th>Adjustment for confounders (^b, c), heavy smoking and blood-Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both eyes</td>
<td>−2.39 (0.78)</td>
<td>−2.30 (0.81)</td>
<td>−2.30 (0.79)</td>
<td>−2.15 (0.82)</td>
</tr>
<tr>
<td>Best eye</td>
<td>−1.20 (0.84)</td>
<td>−0.81 (0.87)</td>
<td>−1.11 (0.85)</td>
<td>−0.66 (0.88)</td>
</tr>
<tr>
<td>Worst eye</td>
<td>−1.95 (0.96)</td>
<td>−1.47 (0.99)</td>
<td>−1.85 (0.97)</td>
<td>−1.27 (1.00)</td>
</tr>
</tbody>
</table>

\(a\) Logarithmically transformed CCI values for best, worst and both eye(s) represent the dependent variables in the multiple linear regression model.

\(b\) Confounders included gender and age.

\(c\) \(\beta\)-values \(\times 10^{3}\).
involve impairment of monoaminergic neurotransmitter systems. Other studies suggest that platelet MAO-B is associated with other aspects of the visual system, including visual illusion strength [37] and visual evoked potentials [36]. In the present study, participants classified as dyschromatopic displayed significantly lower MAO-B activity, when compared to those with normal colour vision. The majority of participants with dyschromatopsia had impairments in the blue-yellow range \((n=54)\), while 25 participants had a combination of red-green and blue-yellow impairment. The high prevalence of dyschromatopsia is most likely due to the age of this cohort, as colour vision is known to decreases with age [27]. Previous reports have suggested that platelet MAO-B activity also increases with age [8]; however, we did not observe this association [52]. Simple correlations using theColour Confusion Index (CCI) scores were also associated with MAO-B activity, as participants with lower enzyme levels had increased acquired colour vision loss. In addition to age, gender is an important confounder that must be addressed. In this study, women had superior colour vision and higher platelet MAO-B activity than men. However, associations between MAO-B and CCI appeared to be independent of gender, as negative correlations were observed separately in both men and women. Multiple linear regression analysis also showed that this association was significant after adjusting for the effects of age and gender, but only when the worst and both eye(s) were used. The relationship between MAO-B and CCI using the best eye was not significant after adjusting for age and gender. It is widely known that acquired dyschromatopsia can be mono-lateral [57], and this may explain why the strength of the associations was dependent on the eye being tested.

The monoaminergic system plays an important role in visual processes as the cellular layers of the primary visual cortex are innervated by both serotonergic and noradrenergic neurons [42]. Dopamine and its receptors have been localized to the retina of vertebrates and have been shown to be important for colour vision function [21,22,41]. The mechanism for acquired colour vision loss is thought to occur through either altered cone function or disrupted neurotransmitters systems in the retina [28]. Studies have shown that the injection of a dopamine receptor (D1) antagonist, SKF38393, or a dopaminergic neurotoxin (6-OH-dopamine) into the vitreous of the eye impairs colour vision discrimination in goldfish [41]. Similarly, reduced colour vision perception in Parkinson’s disease patients can be reversed following the administration of a dopamine precursor, L-DOPA [10]. This evidence suggests that depletion of dopamine in the CNS is the underlying mechanism of impaired colour vision differentiation in the models examined [22]. However, this simple dopamine depletion mechanism of action does not explain the associations between MAO-B activity and colour vision in the present study, suggesting the possibility of an alternative mechanism. In addition to changes in retinal cells, long-term disruption of MAO in other regions of the visual system, including the optic nerve and visual cortex, may be involved in chemical triggered mechanism of colour vision impairment.

Cigarette smokers have reduced colour vision when compared to non-smokers [23]. It is difficult to identify the biochemical mechanism for this interaction due to the numerous chemicals present in tobacco smoke. However, chemicals from tobacco have been identified to reversibly inhibit both rat brain MAO-A and MAO-B function in vitro [12]. Biochemical and PET (positron emission tomography) studies similarly indicate that heavy smoking results in MAO reduction in platelets, brain and other peripheral regions in humans [4,24,25]. In the present study, heavy smoking (>14 cigarettes/day) was associated with approximately a 25% reduction in platelet MAO-B activity [52]. When adjustments were made for heavy smoking, the association between MAO-B and CCI was substantially weakened with the worst eye only. Heavy smoking did not alter this association when CCI from both eyes was used. The visual cortex plays a key role in analysing and interpreting colour signals, particularly when both are used simultaneously [26]. Therefore, it is difficult to interpret these eye-specific responses given the complexity of colour analysis in the cortex. Moreover, it is possible that the established monocular nature of dyschromatopsia may explain some of these differences [57].

**Fig. 1.** Mean (±S.E.) adjusted CCI scores for subjects in platelet MAO-B activity tertile groups (MAO-B range shown brackets). CCI score was adjusted for the effects of age and gender and data for both, worst and best eye(s) are shown. There were 37 or 38 participants per tertile group. Bars with different letters are significantly different, \(P<0.05\).
While a direct cause–effect relationship can not be established, it appears the association between colour vision and platelet MAO-B can be partially explained by the effects of heavy cigarette smoking.

Hg compounds are neurotoxic and exposure to methylmercury (MeHg) is well known to damage the visual cortex [53]. Studies performed in adults showed that visual function, including colour discrimination performance, was impaired by MeHg from dietary fish consumption [35,39]. Similarly, relationships were observed in industrial workers exposed to Hg vapour [14,55], which was shown to be reversible [13]. MeHg is known to inhibit MAO function [15] and we have recently shown that brainstem MAO activity is reduced following a low-level gestational exposure to MeHg in female rats [5]. Given that Hg exposure was also associated with decreased platelet MAO-B activity in the Lake St. Pierre population, it is reasonable to suggest that MAO-B may be a surrogate biomarker of early neurotoxicity for Hg. However, adjustment for the effects of blood-Hg did not alter the association between MAO-B and colour vision. This is likely due to the relatively low Hg exposure range in our studied population (blood-Hg, 0.2–17.0 µg/l). These concentrations are below the lowest observable effect level (behavioural effects and visual impairments) reported for adults in the literature [43].

Results from the current study suggest that in addition to smoking and Hg, other unknown factors may explain the association between MAO-B and colour vision. These unknown factors include genetic variations as well as other environmental factors. Although not observed in the current population, heavy alcohol consumption [40] and industrial exposure to styrene [11] have been previously shown to reduce colour vision. These neurotoxic compounds, in addition to Hg and tobacco smoke, have all been shown to reduce brain and platelet MAO activity in laboratory animals and platelet MAO-B activity in humans [2,16,17,48]. Given that exposure to the above mentioned neurotoxicants are known to cause acquired colour vision loss, there is some circumstantial evidence to suggest that MAO is involved in a common biochemical mechanism of effect on the colour vision system.

In conclusion, these results provide evidence that platelet MAO-B can be used as a biochemical marker for understanding the mechanism of acquired colour vision dysfunction. However, the potential effects of genetic and other environmental factors will need to be further investigated. Moreover, this cross-sectional study only examines a small population in a fishing community with a low range of Hg exposure; it will be important to study whether the same relationship will be observed in the general population. If similar association among neurotoxicant exposure, platelet MAO-B activity and colour vision can be observed in other studies, this biomarker will provide an important tool for future environmental health investigations.

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