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## Review

## Aging and vision

Cynthia Owsley

Department of Ophthalmology, School of Medicine, University of Alabama at Birmingham, Birmingham, AL 35294-0009, USA

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### ABSTRACT

Given the increasing size of the older adult population in many countries, there is a pressing need to identify the nature of aging-related vision impairments, their underlying mechanisms, and how they impact older adults' performance of everyday visual tasks. The results of this research can then be used to develop and evaluate interventions to slow or reverse aging-related declines in vision, thereby improving quality of life. Here we summarize salient developments in research on aging and vision over the past 25 years, focusing on spatial contrast sensitivity, vision under low luminance, temporal sensitivity and motion perception, and visual processing speed.

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## 1. Introduction

Fifty years ago there was little scientific information available on the effect of aging on vision except for that summarized in Robert Weale's now classic book *The Aging Eye* (Weale, 1963). Twenty-five years ago scientists were just beginning to embark on comprehensive programs of research addressing how the aging process impacts various aspects of visual functioning. With the formation of the National Institute on Aging in 1974 as a separate institute within the National Institutes of Health in the US, the scientific spotlight was focused on vision and the aging process per se, rather than solely on eye conditions and diseases prevalent in older adults (e.g., macular degeneration, glaucoma, cataract). By the late 1970s research increasingly addressed the role of lifespan changes during adulthood on visual function and task performance and the mechanisms underlying these aging-related changes. The initial years of this formative period were marked by a symposium entitled "Aging and Human Visual Function" in 1980 sponsored by the Committee on Vision of the National Research Council and convened at the National Academy of Sciences in Washington DC. The symposium, which helped to "jump-start" the field, was later published in an edited book (Sekuler, Kline, & Dismukes, 1982) that served as one of the first reference volumes for the field. Many other overviews have appeared in the ensuing 25 years, and the reader is referred to these for additional perspectives on the field (e.g., Faubert, 2002; Jackson, Owsley, & Curcio, 2002; Kline & Schieber, 1985; Ordy, Wengenack, & Dunlap, 1991; Owsley & Sloane, 1990; Sekuler & Sekuler, 2000a; Sekuler & Sekuler, 2000b; Spear, 1993; Weale, 1982a; Weale, 1986; Werner, Peterzell, & Scheetz, 1990; Werner, Scheffrin, & Bradley, 2010).

Research on vision and aging is essential for several reasons. The percentage of the population in the US and many other countries over age 60 is increasing, and thus eye conditions, diseases, and vision impairments associated with aging represent a larger segment of our societal health challenge on a population basis than in previous decades. Thus, there is a pressing need to identify the prevalence and incidence of various aging-related vision impairments in populations, the mechanisms underlying these impairments, and how they impact older adults' performance of everyday visual tasks. The results of this research can then be used to develop and evaluate interventions to slow or reverse aging-related declines in vision, thereby improving quality of life. Research programs on how vision changes during the course of adulthood can also provide information about biomarkers or risk factors for the incident development of serious eye diseases and conditions common in late adulthood. For example, are there aging-related psychophysical deficits in certain aspects of visual function that are exacerbated in older adults who eventually develop serious eye diseases (e.g., age-related macular degeneration (AMD), glaucoma)? Vulnerabilities in visual function in late life can potentially reveal breakdowns in the visual system that are early markers or signs that disease development is likely. It is also important to know what biological (e.g., neural, optical) and environmental (e.g., smoking, dietary) characteristics differentiate between those older adults who "age well" (e.g., lose little to no visual sensitivity as they age) versus those who do not (e.g., experience substantial threshold elevations). Finally, our understanding of basic visual processes in general benefits from discoveries on older adults' visual capabilities in that the most comprehensive theoretical frameworks and models of vision must account for visual changes over the lifespan.

A methodological challenge in defining normal aging needs to be mentioned at the outset. Many structural and physiological changes

E-mail address: [owsley@uab.edu](mailto:owsley@uab.edu)

in the eye during aging often lie on the same continuum of those changes due to disease. For example, during adulthood the density of the crystalline lens increases (Xu, Pokorny, & Smith, 1997); at what point is it considered cataract? Along similar lines, drusen appear in many older retinas (Vinding, 1990); what characteristics of drusen formation lead to a diagnosis of AMD? Because of the continuum of aging and disease it is important for studies purportedly studying aging to use, whenever possible, explicit inclusion and exclusion criteria that make reference to scales and/or clearly defined characteristics. Stating that the eye studied has been designated as “clinically normal” by an ophthalmologist or optometrist may not be adequate. This is because what one ophthalmologist or optometrist considers clinically normal can differ from one clinician to the next. Eligibility criteria based on retinal fundus grading systems (Age-Related Eye Disease Study Group, 2005) or lens grading systems (Chylack et al., 1993), or other more objective or standardized ways for measuring lens density and scatter are preferred in determining eligibility for recruitment into a study. Clear delineation of these criteria also facilitates comparison of results across studies. The good news is that an increasing number of investigators over the past 25 years are using more objective criteria in defining “normal aging” especially for retinal health (Dimitrov, Guymer, Zele, Anderson, & Vingrys, 2008; Elliott & Werner, 2010; Owsley et al., 2006), yet there are still many studies that rely on clinical judgment alone, self-report and/or a visual acuity cutpoint (e.g., 20/25) to define normal aging. We have already discussed the potential problem with relying on clinical judgment as a case definition of normal aging. Self-report is even more problematic since older adults are often unaware of whether they have early forms of conditions or diseases, which theoretically could be impacting their visual function. About half of older adults do not seek annual comprehensive eye care when conditions might be diagnosed (McGwin, Khoury, Cross, & Owsley, 2010), so many older adults are not in the position to be made aware if they have eye conditions or diseases. The problem with relying on good visual acuity to define normal retinal aging is that good spatial resolution does not necessarily mean that the retina is free of disease; for example, good visual acuity can be retained even though AMD or primary open angle glaucoma is present. Yet these conditions can impact other aspects of retinal function, and thus impact higher-order visual processing as well. Studies not using more objective and standardized criteria for normal retinal health must be viewed in light of the potential confounding effects of disease in interpreting thresholds.

The purpose of this article is not to provide a comprehensive up-to-date overview of the broad field of vision and aging; the topic is simply too broad for an article of this type. As mentioned above, there are several summaries of the literature that when taken together provide a comprehensive overview of our current understanding of vision phenomena in later adulthood (e.g., Jackson et al., 2002; Sekuler & Sekuler, 2000b; Werner et al., 2010). Rather, our purpose here is to identify areas rather intensively studied since 1985, summarize and critically review the current state of knowledge, and discuss the unresolved or new questions emerging from this body of work. Our focus is on studies of visual function as psychophysically measured. The reader will no doubt note that there are many aging and vision phenomena not addressed here, and their omission by no means signifies that the author believes they are unimportant. The topics below were selected because from this author’s perspective they seemed the most salient.

## 2. Spatial contrast sensitivity

The processing of spatial contrast is a fundamental building block of pattern vision. Therefore, it is not surprising that one of

the first questions tackled by vision researchers interested in the aging process was how aging impacts spatial contrast sensitivity. As we will see below, research on this issue has continued into the present. In all the research discussed here, refractive error (including that from presbyopia) is removed as a potential contributor to contrast sensitivity loss by ensuring that observers are refracted for the target distance.

By the end of the 1980s several studies established that older adults have impaired contrast sensitivity under photopic conditions at intermediate and high spatial frequencies, with the magnitude of deficit increasing with increasing spatial frequency (Derefeldt, Lennerstrand, & Lundh, 1979; Elliott, Whitaker, & MacVeigh, 1990; Kline, Schieber, Abusamra, & Coyne, 1983; Owsley, Sekuler, & Siemsen, 1983; Tulunay-Keese, Ver Hoeve, & Terkla-McGrane, 1988). Studies differ in the reported magnitude of this deficit undoubtedly stemming from the variations in lens density and retinal health in the samples used across studies. As mentioned earlier, cataract and AMD themselves can cause contrast sensitivity deficits even in their early forms (Elliott, Gilchrist, & Whitaker, 1989; Kleiner, Enger, Alexander, & Fine, 1988; Rubin, Adamsons, & Stark, 1993); the first studies on aging and contrast sensitivity did not use more objective and standardized criteria for lens and retina in determining participant eligibility. Thus, for example, some study samples undoubtedly had older adults with more lens opacity than other studies, which could lead to differences among studies in the magnitude of psychophysical impairments reported. Thus, efforts to resolve why one study found a larger young-old difference than the other are bound to be difficult if not futile.

Older adults’ spatial contrast sensitivity deficits are present even when criterion-free methods of threshold estimation are used (Elliott et al., 1990; Higgins, Jaffe, Caruso, & deMonasterio, 1988). Impairment in lower spatial frequency sensitivity seems to be spared under photopic conditions. However, if low spatial frequency targets (e.g., 0.5 cycles/degree) are presented with rapid temporal modulation, the sensitivity enhancement characteristic of younger adults is attenuated in older adults (Elliott et al., 1990; Habak & Faubert, 2000; Kline, 1987; Owsley et al., 1983; Sloane, Owsley, & Jackson, 1988). This finding is consistent with the literature on aging and temporal sensitivity showing that older adults exhibit sensitivity loss for flicker that is accentuated as temporal frequency increases (Kim & Mayer, 1994; Tyler, 1989).

Retinal illuminance in older eyes is reduced due to pupillary miosis (Loewenfeld, 1979) and the increased density of the crystalline lens (Pokorny, Smith, & Lutze, 1987; Said & Weale, 1959; Weale, 1961). There is also increased intraocular light scatter and increased optical aberrations in the aging eye (Artal, Guirao, Berrio, Piers, & Norrby, 2003; Glasser & Campbell, 1998) that can reduce image contrast. Research has indicated that optical characteristics of older eyes are largely responsible for older adults’ spatial contrast sensitivity deficits at photopic light levels. When the optical performance of the human eye was compared for younger and older adults using a double-pass apparatus to measure the modulation transfer function, there were lower values of modulation in the older eyes (Artal, Ferro, Miranda, & Navarro, 1993; Guirao et al., 1999). This lower modulation difference was similar to the psychophysically measured loss in spatial contrast sensitivity in older adults as compared to younger adults from previous work (Owsley et al., 1983). That older adults’ loss in spatial contrast sensitivity under photopic conditions is largely optical in origin was also established through psychophysical studies using laser interferometry to bypass the optics of the eye in generating targets on the retina (see Burton, Owsley, and Sloane (1993) for a critical review of this literature). These studies found that older adults either exhibited no (Dressler & Rassow, 1981; Kayazawa, Yamamoto, & Itoi, 1981) or a small yet statistically significant loss in contrast

sensitivity (0.1–0.2 log units) (Burton et al., 1993) when the interference fringe targets are utilized. This 0.1–0.2 loss in “neural” contrast sensitivity accounted for less than half of older adults’ photopic contrast sensitivity loss at higher frequencies when sensitivity was measured using conventional viewing techniques where the optics are not bypassed (Derefeldt et al., 1979; Kline et al., 1983; Owsley et al., 1983; Tulunay-Keeseey et al., 1988), implying that optical factors make a major contribution to spatial contrast sensitivity deficits in older adults at photopic levels (Fig. 1).

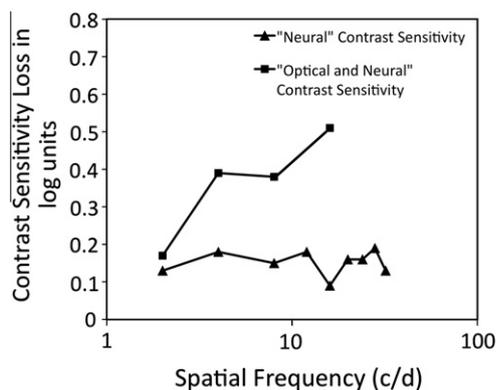
However, other psychophysical studies, while acknowledging that optical changes in the aged eye contribute to aging-related contrast sensitivity deficits, stress that neural factors play a significant role. One study using laser interferometry (Elliott, 1987) reported a neural contribution to older adults’ spatial contrast sensitivity loss that accounted for about half of older adults’ impairment. But this loss occurred only at the highest spatial frequency tested (16.5 cycles/degree). In a recent study (Elliott et al., 2009), adaptive optics (AO) were used to correct monochromatic higher-order aberrations when measuring older adults’ spatial contrast sensitivity; the basic experimental logic was that if older adults’ contrast sensitivity loss disappeared, this would constitute evidence that optical factors, specifically higher-order aberrations, underlie older adults’ deficit in photopic spatial vision. Results were that on average older adults’ spatial contrast sensitivity with AO improved, but not to the level of younger adults’ sensitivity level when they had no AO compensation. As suggested by the authors, these results might imply neural factors play a role in older adults’ loss in photopic contrast sensitivity, but they could also indicate that optical variables other than monochromatic aberrations (e.g., increased lens density and light scatter) could be playing a larger role once AO corrections are introduced.

Although in our view strong evidence that neural factors have a sizeable influence on older adults’ photopic contrast sensitivity is lacking, it is important to point that Curcio and Drucker (1993) have shown that ganglion cell density was reduced by 25% in the central 11° of retina in donor eyes from older adults, compared to eyes from young adults; Gao and Hollyfield (1992) also report aging-related ganglion cell density declines although less steep than those reported by Curcio and Drucker (1993). However, loss in neural elements does not necessarily correspond to functional deficits as measured psychophysically. Furthermore, although Curcio and Drucker (1993) and colleagues reported decreased

aging-related ganglion cell density, Curcio and colleagues found a remarkable stability throughout adulthood in cone photoreceptor density throughout the retina including the fovea and the macula (Curcio, Millican, Allen, & Kalina, 1993). With respect to aging-related structural changes later in the visual pathways, research on post-retinal visual pathways has not been extensive, and what does exist has been in animal models. In old monkeys decreased neuron density in both the magnocellular and parvocellular layers of the lateral geniculate nucleus (LGN) was reported (Ahmad & Spear, 1993), but this was not due to a loss in neurons. Rather, it stemmed from a minor decrease in the number of neurons coupled with an increase in LGN volume. Thus, from at least one study, there is no evidence that LGN deterioration is contributing to spatial vision loss in older adults. Aging effects on visual cortical cells have been more noteworthy. Single-unit recording studies in V1 performed on old monkeys found that cortical neurons exhibited lower optimal spatial and temporal frequencies, less sensitivity to contrast, lower spatial resolution, and lower higher temporal frequency cut-offs as compared to recordings in young monkeys (Yang et al., 2008; Zhang et al., 2008). In addition, these cells in older monkeys were accompanied by increased spontaneous, visually-driven activity and decreased signal-to-noise ratio. Many of these effects were more pronounced in area V2 as compared to V1 (Wang, Zhou, Ma, & Leventhal, 2005). Results from monkey models suggest that it is conceivable that changes in cortical cells in V1 and V2 could be producing spatial contrast sensitivity deficits observed in older adult humans, however, these studies do not confirm that this is the case.

To summarize, 25 years of research on spatial contrast sensitivity under photopic conditions in older adults converges on the following points. For older adults, spatial contrast sensitivity loss under photopic conditions increases with increasing spatial frequency. Low spatial frequency sensitivity at photopic levels is not impacted by aging, or only minimally, when targets are presented at low temporal frequencies. However, sensitivity for low spatial frequencies is impaired under rapid temporal modulation, which probably stems from the reduced temporal sensitivity of the aging visual system. Optical characteristics of the aged eye reduce spatial contrast sensitivity. These factors include reduced retinal illuminance (either from pupillary miosis, increased lens density, or both), increased intraocular light scatter, and increased aberrations. (Not discussed above, it must be acknowledged that pupillary miosis can also have a positive impact on older adults’ spatial vision by reducing optical aberration.) Although some psychophysical studies provide data implying there is a neural contribution, these studies do not provide definitive evidence that neural deterioration in the visual pathways are practically significant contributions to older adults’ loss in spatial contrast sensitivity. Reduced ganglion cell density in older donor retina from human studies and deleterious changes in the spatial and temporal response properties of visual cortical neurons in old monkey models suggest that neural deterioration in the primary visual pathway could contribute to older adults’ spatial contrast sensitivity loss. However, it remains to be determined whether these structural changes actually do impact photopic spatial vision in older humans.

At this juncture it is constructive to take a look at the magnitude of the threshold elevation effect in older adults for which the field has persistently been searching for a cause for 25 years. Is the young-old difference in contrast sensitivity of such a magnitude that it is practically or theoretically significant? The difference between contrast sensitivity for 20-year-olds and 70-year-olds at approximately 8 cycles/degree ranges from 0.2 to 0.57 log units, depending on which study is used as a source (Elliott, 1987; Elliott et al., 1990; Elliott et al., 2009; Kline et al., 1983; Owsley et al., 1983), with the average young vs. old sensitivity difference across



**Fig. 1.** Adapted from Burton et al. (1993). Comparison of older adults’ loss in photopic spatial contrast sensitivity using interference fringes (labeled “Neural” Contrast Sensitivity) and older adults’ vision loss in a direct viewing spatial contrast sensitivity task from Owsley et al. (1983) where the optics were not bypassed (“Neural + Optical” Contrast Sensitivity). The contrast sensitivity loss for interference fringes accounts for less than half of the contrast sensitivity loss at higher spatial frequencies in the direct view task. This suggests that older adults’ spatial contrast sensitivity loss under photopic conditions is largely optical in origin.

these studies being approximately 0.3 log units. Is the size of this aging effect sufficient to merit persistent investigation? Granted, it appears to be a repeatable effect as conveyed by the number of studies that show statistically significant young-old differences. But does research on the causes of the effect, i.e., mechanisms, have pressing significance? May be not, for the following reasons. First, as just reviewed, optical mechanisms largely account for the loss. Furthermore, the size of the young versus old difference itself is small, and in some studies, the methods used for estimating threshold may have had a test–retest reliability similar in size to, or exceeding, the effect size. Although an effect of this size can elevate contrast thresholds for real-world targets (Akutsu, Legge, Ross, & Schuebel, 1991; Owsley, Sekuler, & Boldt, 1981), there are a variety of compensatory strategies that older adults can use to increase the visibility of targets, such as moving closer to the target, scanning the target, increasing viewing time, and if illumination is low, by increasing illumination. Thus, a 0.3 log unit loss in contrast sensitivity is not apt to be visually disabling although it may be modestly visually impairing. The primary cause of the optical degradation in the aged eye is likely to be cataractous lens changes. When visibility problems impinge on quality of life and visual task performance, older adults elect cataract surgery, which is covered by health insurance or government health programs in many countries. Therefore, until the time an older adult undergoes cataract surgery, these aging-related contrast reductions may be operative, but in the longer term, they are largely reversible.

None of this is meant to be an argument that we should not seek to improve our understanding of how aging impacts the visual pathways in the brain. The point is that research over the past 25 years taken as a whole makes a reasonable case that that aging-related spatial contrast sensitivity deficits under photopic conditions are largely optical in origin. Ultimately for many older adults this optical deficit is in large part correctable through cataract surgery and intraocular lens insertion. Therefore, it is reasonable to suggest that going forward the field move beyond studies searching for a neural basis of a phenomenon that is largely optical, is small compared to other types of aging-related visual deficits, and lacks serious detrimental significance to older adults in the longer term.

### 3. Scotopic contrast sensitivity

A common visual complaint described by older adults including those in good eye health is difficulty seeing under low illumination or at night. Older adults cite these problems in both surveys and focus groups. For example they report difficulty reading under dim light (Kosnik, Winslow, Kline, Rasinski, & Sekuler, 1988; Mangione et al., 1998; Owsley, McGwin, Scilley, & Kallies, 2006) and modify their behavior by avoiding night driving (Ball et al., 1998; Brabyn, Schneek, Lott, & Haegerström-Portnoy, 2005). Poor vision under reduced light levels and at night in the elderly has been linked to their involvement in motor vehicle collisions and falls (Källstrand-Ericson & Hidingh, 2009; Massie, Campbell, & Williams, 1990; McMurdo, 1991; Mortimer & Fell, 1989). Older adults who report visual difficulty under poor lighting conditions are also those who are more likely to have scotopic sensitivity impairment as determined psychophysically (Owsley, McGwin, Scilley et al., 2006b; Scilley et al., 2002). Over the past 25 years two lines of research have addressed low luminance vision problems in older adults – one focus is on diminished spatial contrast sensitivity under low luminance, which will be discussed here, and the other is slowing in the rate of dark adaptation, which will be discussed in the next section.

Older adults' loss of spatial contrast sensitivity at low luminance levels (scotopic and mesopic) is accentuated compared to

their loss under photopic conditions including a decline in older adults' high spatial frequency cut-off as compared to that for young adults (Scheffrin, Tregear, Harvey, & Werner, 1999; Sloane, Owsley, & Alvarez, 1988; Sloane, Owsley & Jackson, 1988). To gain an appreciation for the disadvantage older adults face under scotopic conditions, consider that young and older adults have the same sensitivity for a 0.5 cycles/degree grating in daylight, whereas under scotopic conditions older adults require on average three times the contrast of younger adults in order to discern the target (Scheffrin et al., 1999) (Fig. 2). There is also evidence that this deficit increases as background adaptation level decreases in the mesopic and scotopic ranges, and the deficit may be the most severe at lower spatial frequencies (Clark, Hardy, Volbrecht, & Werner, 2010; Scheffrin et al., 1999). Like photopic spatial contrast sensitivity deficits, mesopic and scotopic deficits have an optical contribution, but unlike photopic sensitivity, neural factors appear to play a major role in spatial vision loss under mesopic and scotopic conditions. On first analysis, it would seem that decreased rod photoreceptor density and ganglion cell density during the aging process (Curcio et al., 1993; Curcio & Drucker, 1993; Gao & Hollyfield, 1992) are logical candidate mechanisms underlying decreased spatial vision under low luminance in older adults. It could be the case that the loss in density of these neural elements and/or the enlargement of rod inner segments whereby they occupy the space left by necrotic rods (Curcio et al., 1993) increase receptive field center size for ganglion cells. However, psychophysical data have not supported this purported mechanism. Scheffrin, Hauser, and Werner (2004) reasoned that if the retinal ganglion cell receptive field centers that receive rod input are increased in size in the older adult, then Weber-like behavior (e.g., contrast sensitivity increases monotonically with mean luminance level for a spatial target) should theoretically occur at a lower luminance level for older adults as compared to young adults. Yet younger and older adults displayed similar mean luminance levels corresponding to Weber-like behavior onset (Scheffrin et al., 2004), implying that ganglion cell receptive field centers do not get larger during aging.

Other research has demonstrated an enlargement in Ricco's area under scotopic conditions, the largest area over which complete spatial summation holds (Scheffrin, Bieber, McLean, & Werner, 1998; Scheffrin et al., 1999), which cannot be attributed to optical factors. Scheffrin et al. (1998) suggest that this change in spatial summation could be attributable to a change in the relative sensitivities of spatially selective channels under scotopic conditions or

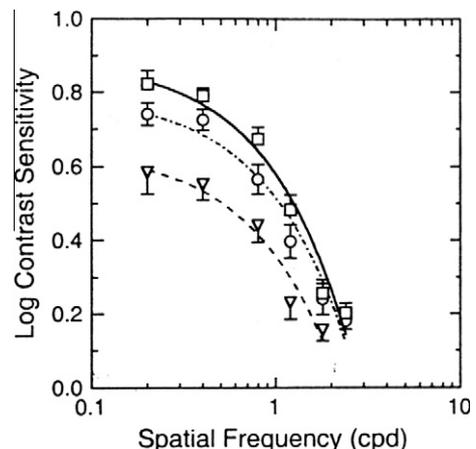


Fig. 2. From Scheffrin et al. (1999). Average scotopic spatial contrast sensitivity for three age groups. Squares signify 20–40-year-olds, circles 41–60-year-olds, and triangles 61–88-year-olds. Note that these losses are sizeable at low spatial frequencies and cannot be attributed to optical factors, suggesting a neural origin.

possibly synaptic re-wiring at either the retinal or cortical levels. It remains to be determined what retinal and cortical mechanisms underlie older adults' impairment in scotopic contrast sensitivity, and to determine whether this scotopic dysfunction is modifiable through intervention.

#### 4. Dark adaptation

As far back as the 1940s it has been known that older adults have decreased light sensitivity in the dark and this deficit is larger than for photopic thresholds (Birren & Shock, 1950; Gunkel & Gouras, 1963; Jackson & Owsley, 2000; Jackson, Owsley, Cordle, & Finley, 1998; McFarland, Domey, Warren, & Ward, 1960; Robertson & Yudin, 1944; Steven, 1946; Sturr, Zhang, Taub, Hannon, & Jackowski, 1997; Weale, 1982b). Although increased optical density of the aged crystalline lens and pupillary miosis contribute to their scotopic threshold elevation (increasing threshold by about a 0.10–0.15 log units), more recently it has been established that these factors are not primarily responsible for this sensitivity loss, with about a half log unit elevation in threshold or more remaining after these factors are taken into account (Jackson et al., 1998; Sturr et al., 1997). Only in the past decade or so has research addressed potential neural mechanisms that underlie the loss. By age 60–70 years old, the density of rod photoreceptors decreases dramatically in the peri-macula as indicated by studies on donor retinas (Curcio et al., 1993; Gao & Hollyfield, 1992). However, scotopic sensitivity loss in older adults occurs in retinal areas where there is negligible rod loss and is not accentuated in the areas of heightened rod loss (Jackson et al., 1998), suggesting that rod loss by itself cannot account for older adults' sensitivity impairment in the dark. Furthermore, there is little change in the amount of rod photopigment, rhodopsin, throughout adulthood (Liem, Keunen, van Norren, & van de Kraats, 1991; Plantner, Barbour, & Kean, 1988; van Kuijk, Lewis, Buck, Parker, & Klinger, 1991).

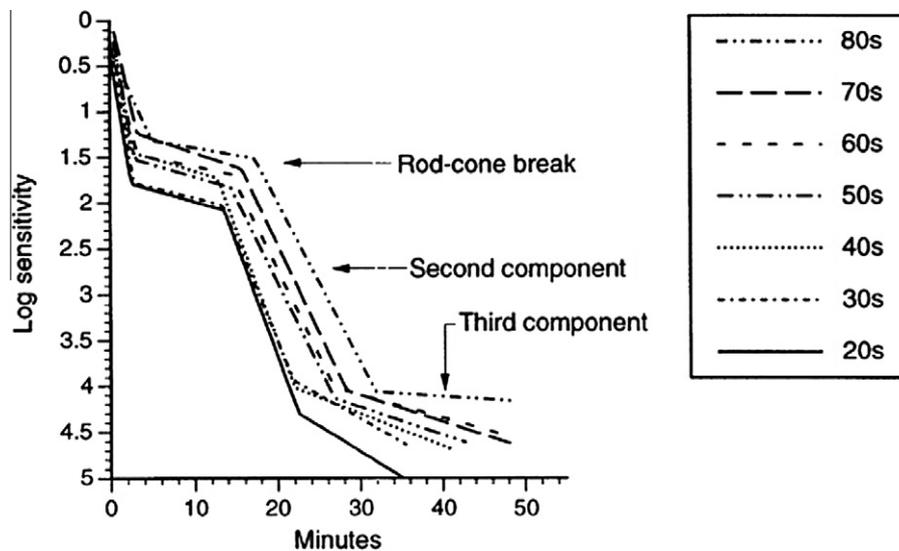
An alternative explanation for older adults' scotopic sensitivity loss is that the visual cycle, the biochemical pathway responsible for rhodopsin regeneration, is perturbed with age. The visual cycle includes the production of 11-*cis*-retinal from retinoid and the subsequent regeneration of rhodopsin. Slowing of the visual cycle results in a prolongation of dark adaptation kinetics. Psychophysical dark adaptometry techniques can estimate the time constants associated with the visual cycle by measuring the recovery of light sensitivity after exposing the photopigment to an intense light that bleaches the photopigment (Alpern, 1971; Barlow, 1972; Hecht, Haig, & Chase, 1937; Lamb & Pugh, 2004; Rushton & Powell, 1972). Jackson, Owsley, and McGwin (1999) studied rod-mediated dark adaptation in older adults overcoming the methodological shortcomings of the earlier work. They found that older adults experience substantial delays in adapting to darkness (Fig. 3). Specifically, they found that older adults exhibited an increase in the time constant for the second and third components of rod-mediated dark adaptation (Lamb & Pugh, 2004; Leibrock, Reuter, & Lamb, 1998), indicating slowing in rhodopsin degeneration. These psychophysical results, later replicated (Owsley, McGwin, Jackson, Kallies, & Clark, 2007), are consistent with results obtained by rod densitometry (Liem et al., 1991). From a practical everyday standpoint, the problems faced by older adults in adjusting to darkness are non-trivial (e.g., adjusting to dark indoor environment after being outside on sunny day; searching for object in dark closet or drawer). The time taken for 70-year-olds to reach pre-bleach light sensitivity is over 10 min longer than for those in their 20s.

What might be contributing to slowed rhodopsin regeneration in older adults? Photoreceptor function and survival are critically dependent on the retinal pigment epithelium (RPE) and Bruch's membrane to regulate the transport of nutrients, fluid, ions, and

metabolites to and from the subretinal space (Bok, 1985). During aging there are changes in the RPE–Bruch's membrane complex such as progressive thickening of Bruch's membrane (Bird, 1992; Feeney-Burns & Ellersieck, 1985; Newsome, Huh, & Green, 1987), accumulation of extracellular material between the RPE and Bruch's membrane (Curcio, Millican, Bailey, & Kruth, 2001; Pauleikhoff, Harper, Marshall, & Bird, 1990), reduced hydraulic conductivity of Bruch's membrane (Starita, Hussain, Pagliarini, & Marshall, 1996), and changes in the structure of RPE cells (Kornzweig, 1979). These changes could compromise metabolic exchange by causing a diffusion barrier between the choroid and photoreceptors, causing a localized scarcity of vitamin A, critical for rod photoreceptor function (Dowling & Wald, 1958; Kemp, Jacobson, & Faulkner, 1988). Psychophysical data are consistent with this explanation. In a recent study (Owsley, McGwin, Jackson et al., 2006) older adults' dark adaptation was measured before and after they received a 30-day, high-dose course of retinol (pre-formed vitamin A). Older adults receiving the retinol course had rod-mediated sensitivity recovery that was faster as compared to a placebo-control group. The responsiveness of rod-mediated dark adaptation to a short course of high-dose retinol is consistent with the hypothesis that depositions and other structural changes in the RPE/Bruch's membrane complex in aging caused a diffusion barrier that disrupts normal metabolic exchange, leading to a local shortage of vitamin A. Although this study does not provide direct evidence of an *in vivo* localized nutritional deficiency, it does highlight a possible pathway by which rod dysfunction and degeneration could occur during aging, and why dark adaptation is delayed in the elderly. These data cannot inform us of the possible site of the dysfunction that limits the availability of retinoids necessary for visual sensitivity recovery in older adults. As mentioned above, increased systemic vitamin A concentrations may force additional vitamin A across Bruch's membrane into the RPE cells, via mass action, or, alternatively, increased levels of vitamin A may overcome possible impaired transport between the RPE cells and rod outer segments.

Slowing in cone-mediated dark adaptation in the fovea has also been reported for persons in their 60s and 70s (Coile & Baker, 1992). The slowing of sensitivity recovery was closely related to pigment regeneration rate implying that the latter contributes to older adults' cone-mediated delay in sensitivity recovery (see also Keunen, Norren, & Meel, 1987; Kilbride, Hutman, Fishman, & Read, 1986; van Norren & van Meel, 1985). However, another study (Eisner, Fleming, Klein, & Mauldin, 1987) found that the cone-mediated time constant for sensitivity recovery was unchanged between the 60s and the 80s, suggesting potential individual differences among older adults, possibly a leveling off of aging-related changes after the 70s, or/and methodological differences among studies (e.g., bleach magnitude).

Rod-mediated dark adaptation delays are also a hallmark of early AMD (Haimovici, Owens, Fitzke, & Bird, 2002; Owsley, Jackson, White, Feist, & Edwards, 2001; Owsley et al., 2007; Steinmetz, Haimovici, Jubb, Fitzke, & Bird, 1993). Histopathological studies on human donor retinas with AMD indicate a predilection for parafoveal loss of rod photoreceptors over cones in early non-exudative disease, with foveal cone loss not being observed until later in AMD progression (Curcio, Medeiros, & Millican, 1996; Medeiros & Curcio, 2001). Although both rods and cones in the parafovea degenerate in AMD, rod loss precedes and is more severe than cone loss in most donor retinas evaluated, and even in the exudative form, there is greater retention of cones versus rods. These findings have led to the question as to whether during the course of aging, accentuated slowing in the recovery during rod-mediated dark adaptation is a marker for those older adults at high-risk for the eventual development of AMD (Jackson, Curcio, Sloan, & Owsley, 2004; Jackson et al., 2002). Prospective data are



**Fig. 3.** From Jackson et al. (1999). Dark adaptation as a function of decade. Arrows label the portion of the function representing the rod-cone break and the second and third components of rod-mediated dark adaptation. Note that the funcs shift to the right with increasing decade, indicating a slowing in the rate of dark adaptation during aging.

needed to establish whether rod-delays pre-date the emergence of the disease. If so, this line of research not only sheds light on mechanisms of early AMD pathogenesis, but is also relevant for clinical research. For example, a psychophysical biomarker could be used to identify older adults at high-risk for early AMD and thus most appropriate for enrollment in clinical trials evaluating treatments for early AMD (Jackson & Edwards, 2008). In addition, a functional test could serve as an outcome measure for evaluating the impact of treatment interventions, particularly in early AMD where visual acuity remains good and thus is insensitive indicator of efficacy or proof of concept for early disease or preventative interventions (Csaky, Richman, & Ferris, 2008). Cone-mediated dark adaptation delays have also been reported for older adults with early AMD or for those at high-risk for early AMD because of fundus appearance or exudative disease in the fellow eye (Binns & Margrain, 2007; Dimitrov et al., 2008; Eisner, Klein, Zilis, & Watkins, 1992; Sunness et al., 1997; Sunness et al., 2008). Thus, prospective work to identify potential aging-related biomarkers for AMD should also address whether cone dysfunction in older adults pre-dates the emergence of AMD.

### 5. Processing of time-varying targets

Many older adults have deficits in visually processing temporal information, which can hamper the visual performance of everyday tasks. For example, slowed visual processing speed in older adults increases their risk for motor vehicle collision involvement even in the absence of impaired visual acuity, contrast sensitivity, and visual field sensitivity (Cross et al., 2009; Owsley et al., 1998; Rubin et al., 2007), increases the time it takes them to complete visual tasks of everyday living such as finding an item on a shelf or reading a prescription bottle (Ball et al., 2002; Edwards et al., 2002; Owsley, McGwin, Sloane, Stalvey, & Wells, 2001), and is associated with problems with ambulatory mobility (Owsley & McGwin, 2004). Older drivers with elevated thresholds in a coherent motion task had difficulties with detecting signs and hazards on the road, took longer to drive a route, and had worse performance evaluations as assessed by raters specialized in on-road assessment (Wood, 2002; Wood, Anstey, Kerr, Lacherez, & Lord, 2008). Those older adults who reported more difficulty with certain driving maneuvers were also more likely to have impaired performance on speed discrimination, and estimates of the direc-

tion of heading and time to collision (Raghuram & Lakshminarayanan, 2006). In this section, we review what we have learned in the past 25 years about how aging impacts temporal sensitivity and motion perception, and in the next section, we address aging and slowed visual processing speed.

It has been known for over 50 years that critical flicker fusion (CFF) is decreased in older adults (Coppinger, 1955; McFarland, Warren, & Karis, 1958; Misiak, 1947). But only in the past 25 years has research comprehensively explored older adults' problems in processing time-varying targets. Temporal contrast sensitivity is impaired in older adults, more so at higher temporal frequencies than at lower. The deficit cannot be wholly accounted for by optical characteristics of the aged eye (Kim & Mayer, 1994; Mayer, Kim, Svingos, & Glucs, 1988; Tyler, 1989; Wright & Drasdo, 1985). Measurements of the impulse response function (related to the temporal contrast sensitivity function) in older adults using a double-pulse method showed that the amplitude of both the excitatory and inhibitory responses is reduced in later adulthood (Shinomori & Werner, 2003). There were also individual differences; some older adults age  $\geq 70$  years old showed an inhibitory phase in the response amplitude while others did not. Although a retinal locus for these changes is likely to be contributory, post-retinal sites(s) may also play a role (Gerth, Sutter, & Werner, 2003).

Ball and Sekuler (1986) were the first to demonstrate impaired motion perception in older adults that cannot be accounted for by spatial vision or cognitive differences between young and older observers. Specifically, they found that older adults were less able to discriminate between two directions of motion as depicted by random dot displays as compared to young adults. Since this report 25 years ago, many studies have probed motion-processing deficits in the elderly. For example, elevations in both detection and discrimination thresholds in a variety of motion tasks have been documented for older adults suggesting a reduced sensitivity to motion and impaired abilities to identify the direction of movement and differentiate differences in speed (Anderson & Atchley, 1995; Atchley & Anderson, 1998; Norman, Ross, Hawkes, & Long, 2003; Raghuram, Lakshminarayanan, & Khanna, 2005; Snowden & Kavanagh, 2006; Tran, Silverman, Zimmerman, & Feldon, 1998; Trick & Silverman, 1991). There is preliminary evidence that these deficits do not appear gradually during the course of adulthood, but rather, may emerge in late adulthood around the 70s (Bennett, Sekuler, & Sekuler, 2007). Furthermore, increasing the duration of

the stimulus allows some older adults to improve their performance to the level of young adults (Bennett et al., 2007; Raghuram et al., 2005), which suggests that extending viewing time for older adults in a real-world performance task dependent on motion processing may at least partly compensate for deficits.

Bennett et al. (2007) found deficits in both detection sensitivity and direction discrimination for coherent global flow when measured in the same older adults (Fig. 4). They found that direction judgments were more difficult for older adults than mere detection, persisted even after adjustment for aging-related motion sensitivity differences, and that there was larger divergence between a percept of global flow and perceiving the actual direction of flow among older adults than among young adults. A higher level of additive intrinsic noise and a wider bandwidth for directionally tuned cortical mechanisms was needed to model the older adult

data as compared to modeling the young data. These findings in older adults are consistent with recent neurophysiological studies in old monkeys finding that, compared to young monkeys, neurons in the primary visual cortex of old monkeys had increased internal noise and reduced orientation and directional selectivity (Schmolesky, Wang, Pu, & Leventhal, 2000; Wang et al., 2005). The cells' direction and orientation selectivity could be improved by local administration of GABA and GABA agonists, with some cells improving to the level of cells from young monkeys (Leventhal, Wang, Pu, Zhou, & Ma, 2003). These intriguing results not only provide evidence of a decreased inhibition in older visual cortex but that it is also reversible, at least in an animal model.

Psychophysical measurements of motion perception imply that there is indeed decreased visual cortical inhibition in older adults. Betts, Taylor, Sekuler, and Bennett (2005) used a task previously developed (Tadin, Lappin, Gilroy, & Blake, 2003) to study surround inhibition when processing a high-contrast moving pattern. Tadin et al. (2003) found that young adults needed longer stimulus durations to discriminate the direction of a moving high-contrast grating, as compared to the shorter durations required for the task when a low contrast grating was used. This effect was interpreted as evidence of center-surround spatial suppression whereby a neuron's response to a high-contrast stimulus is inhibited when the stimulus boundaries extends beyond its receptive field (Angelucci & Bullier, 2003; Sceniak, Ringach, Hawken, & Shapley, 1999). When Betts et al. (2005) measured the performance of older adults in this task, they found that older adults needed briefer, not longer, durations to discrimination motion direction for large, high-contrast gratings than did young adults. This pattern of findings suggests the existence of decreased visual cortical inhibition in the older adult cortex for motion-sensitive neurons. Following from Leventhal's work in the senescent monkey model (Leventhal et al., 2003), the candidate mechanism for this decreased suppression could be reduced GABAergic functioning in the older brain. Although the Betts et al. study illustrates conditions where older adults could be characterized as having a superior motion perception ability, there also could be some adverse perceptual consequences to this phenomenon (Tadin & Blake, 2005). Older adults displaying this motion perception advantage may have difficulty in ignoring background motion since it is a large (often high-contrast) stimulus field, while at the same time having a weaker sensitivity to smaller, moving figures. This is a common stimulus condition while driving where the "figure" could be an obstacle (e.g., another car or pedestrian), and the "ground" is the rest of the roadway environment. This perceptual characteristic in older adults might contribute to driving problems faced by some older adults (e.g., turning left across oncoming traffic), an issue worthy of further investigation.

Several studies have found an aging-related deficit in the ability to use visual motion to perceive aspects of a 3-d world and spatial layout. For example, older adults have exhibited deficits in using motion to extract self-motion information (Warren, Blackwell, & Morris, 1989), to perceive shape (Blake, Rizzo, & McEvoy, 2008; Norman, Bartholomew, & Burton, 2008; Norman, Clayton, Shular, & Thompson, 2004; Norman, Dawson, & Butler, 2000; Wist, Schrauf, & Ehrenstein, 2000), and to identify collision paths (Andersen, Cisneros, Saidpour, & Atchley, 2000; Anderson & Enriquez, 2006). Under certain stimulus conditions older adults also show deficits in the perception of biological motion (Billino, Bremmer, & Gegenfurtner, 2008; Norman, Payton, Long, & Hawkes, 2004), which may stem from difficulties in efficiently integrating local motion cues with global motion information (Pilz, Bennett, & Sekuler, 2010). All or some of these deficits could be contributing to older adults' difficulties in the performance of everyday tasks such as mobility, driving, visual search, and objective recognition (Ball et al., 2002; Edwards et al., 2002; Owsley & McGwin, 2004;

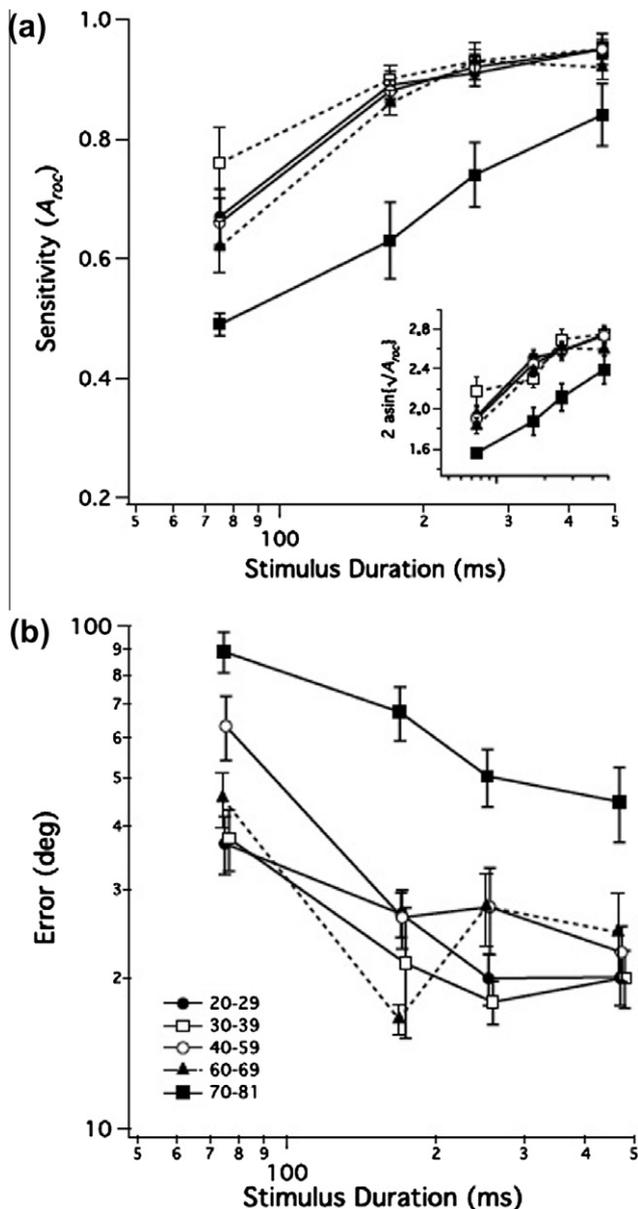


Fig. 4. From Bennett et al. (2007). Panel A shows sensitivity to motion for various age groups as a function of stimulus duration. The inset shows the data after an arcsine transformation. Note that adults  $\geq 70$  years old have impaired motion sensitivity. Panel B shows error magnitude in judging direction of motion (on trials for which motion was detected) for various age groups as a function of stimulus duration. Note that adults  $\geq 70$  years old exhibited larger direction judgment errors.

Owsley, McGwin et al., 2001; Wood et al., 2008). However, research to date has largely ignored an examination of links between aging-related deficits in motion processing and perceiving and acting in a 3-d world, with a few noteworthy exceptions on driving and the visibility of pedestrians in the roadway environment (Raghuram & Lakshminarayanan, 2006; Tyrrell et al., 2009; Wood, 2002; Wood, Tyrrell, & Carberry, 2005; Wood et al., 2008).

## 6. Visual processing speed

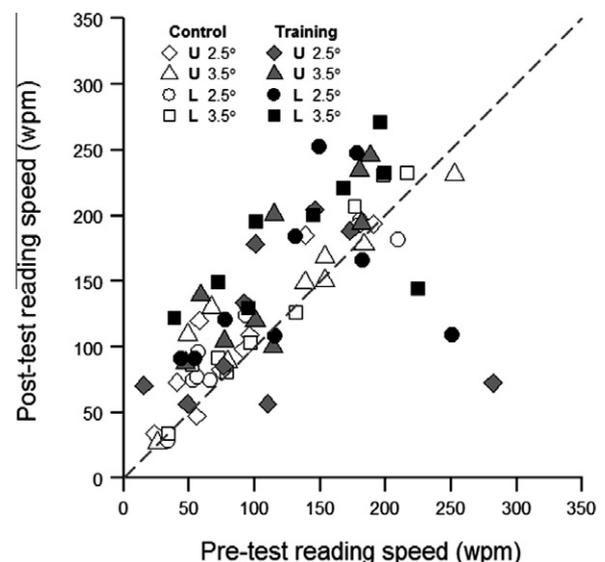
Slowing in visual processing speed is a common characteristic of aging, and has been well established as a phenomenon since the 1970s (Kline & Birren, 1975; Walsh, 1976; Walsh, Williams, & Hertzog, 1979). Many older adults require more time than younger adults to detect, discriminate, recognize, or identify visual targets, and this slowing contributes to higher-order processing problems characteristic of cognitive aging (e.g., associative learning, working memory, inhibition) (Salthouse, 1991; Salthouse, 1993; Salthouse, 1994; Salthouse & Meinz, 1995). These deficits occur even in older adults who do not have conditions that cause dementia (e.g., Alzheimer's disease, cerebrovascular accident). Ball's work has demonstrated that aging-related slowing in visual processing speed is exacerbated by increasing attentional task demands (e.g., divided attention tasks) and by increasing visual clutter (e.g., distracting stimuli) (Ball, Edwards, & Ross, 2007; Ball, Roenker, & Bruni, 1990). That is, in performing laboratory tasks, the display duration needed by many older adults to complete a task under dual task conditions with distracting stimuli is proportionately greater than what is needed by young adults. Much of the work in this field has made use of a task originally described by Ball, Sekuler, and others called the useful field of view task (Ball, Beard, Roenker, Miller, & Griggs, 1988; Ball, Beard, Roenker, Miller, & Griggs, 2006; Ball et al., 1990; Ball et al., 2007; Edwards et al., 2006; Sekuler & Ball, 1986; Sekuler, Bennett, & Mamelak, 2000). The task has been refined over the years. The essential features of the task are that the observer is asked to identify a central target while simultaneously determining the location of a peripheral target, which on some trials is embedded in a field of distractors. Performance is measured by the minimum stimulus duration needed to perform the task (there is no motor response component).

It is important to emphasize that slowed visual processing speed during later adulthood is not inevitable in that there are wide individual differences, with some older adults exhibiting processing speeds like those of young adults, and others having serious slowing (Ball, Owsley, Sloane, Roenker, & Bruni, 1993; Rubin et al., 2007). Unlike other types of visual psychophysical deficits discussed above, a great deal of research has already demonstrated that slowed processing speed in older adults has negative implications for their everyday life. Slowed visual processing in the elderly is associated with increased crash risk (Ball et al., 2006; Cross et al., 2009; Owsley et al., 1998; Rubin et al., 2007), increased fall risk (Sims, Owsley, Allman, Ball, & Smoot, 1998; Vance, Ball, Roenker, Wadley, Edwards, & Cissell, 2006), mobility problems such as transitioning from sitting to standing (Owsley & McGwin, 2004; Riolo, 2003), and increased time needed to complete visual tasks typical of everyday life (Owsley, McGwin et al., 2001; Owsley, Sloane, McGwin, & Ball, 2002). What is particularly promising is that for some older adults, processing speed can be improved, i.e., "speeded up", through practice (Ball et al., 1988; Ball et al., 2002; Edwards et al., 2002; Roenker, Cissell, Ball, Wadley, & Edwards, 2003; Sekuler & Ball, 1986). This training intervention, described in detail elsewhere (Ball et al., 2007), basically consists of computer-based nonverbal exercises that are visually presented very briefly and involve practice in the detection, identification, discrimination, and localization of visual targets. Speed of processing training focuses

on improving the speed of visual search and the ability to perform one or more attentional tasks quickly. During training, the stimulus duration of the visual display in each visual task (e.g., discrimination) is systematically reduced and the tasks are made increasingly more difficult (discrimination task in central vision alone versus discrimination and localization of a peripheral target).

The speed of processing gains by older adults have been shown in one study to be enduring up to 5 years (Willis et al., 2006). Most importantly, intervention evaluations including multi-site randomized trials have demonstrated that faster processing speed in older adults, or prevention of further slowing in processing speed as one ages, enhances several aspects of everyday functioning and health in older adults. Specifically, visual processing speed training led to more efficient completion of everyday visual tasks (less time needed) (Edwards, Wadley, Vance, Roenker, & Ball, 2005; Edwards et al., 2002), reduced motor vehicle collision risk (Ball, Edwards, Ross, & McGwin, in press), improved health-related quality of life (Wolinsky et al., 2006), reduced risk of clinical depression or depressive symptoms (Wolinsky et al., 2009; Wolinsky et al., 2009), and improvements in self-rated health (Wolinsky et al., 2010).

The modifiability of visually processing speed in the context of reading speed in older adults is relevant for understanding whether it is reasonable to expect that older adults with a central scotoma due to AMD can be trained to increase their reading speed in the retinal periphery. The term "visual span" with respect to reading refers to a spatial property of the visual field defined as the number of characters that can be recognized with no eye movement (Legge, Ahn, Klitz, & Luebker, 1997; O'Regan, 1990). The visual span is smaller in the retinal periphery and has been shown to limit reading speed (Legge, Mansfield, & Chung, 2001). Perceptual training can enlarge the visual span in young adults with normal peripheral vision which is accompanied by an increase in reading speed (Chung, Legge, & Cheung, 2004). More recently, this research group (Yu, Cheung, Legge, & Chung, 2010) has extended these findings to older adults with normal vision (Fig. 5). The training benefits were smaller than those observed



**Fig. 5.** From Yu et al. (2010). Scatter plot showing relationship between pre-training reading speed and post-training reading speed for older adults with normal vision. The reading task was performed at 10° in the periphery. Those undergoing training are solid symbols, controls are open symbols. U and L stand for upper and lower visual field, respectively, for location of the test. 2.5° and 3.5° refer to print size. Note that most filled symbols from the training group are above the diagonal indicating improved reading speeds in older adults following training.

for young adults, which may be related to older adults' reduced retention of training gains from session to session (Yu et al., 2010). In light of robust training gains seen in the visual processing speed training studies described above, future work should address whether larger reading speed benefits may be possible if a speed-of-processing training component were incorporated into a perceptual training program to improve reading speed in the periphery.

## 7. Other intriguing issues in vision and aging research

Older adults' threshold elevations for visually complex stimuli, i.e., second-order stimuli, are more accentuated than for simpler stimuli, i.e., first-order stimuli (Faubert & Bellefeuille, 2002; Habak & Faubert, 2000; Habak, Wilkinson, & Wilson, 2009; Tang & Zhou, 2009). In vision research, particularly in the study of motion perception, first- and second-order stimuli have been exploited to improve our understanding of early visual processing, with first-order stimuli defined by luminance modulation, and second-order by features such as contrast, texture, or depth. At least partly different mechanisms underlie the processing of first versus second-order visual stimuli (e.g., Baker, 1999; Lu & Sperling, 2001), and the computational models developed to account for their visual processing are also different (e.g., Chubb & Sperling, 1989; Wilson, Ferrar, & Yo, 1992). Although there are varying reports in the literature, both types of stimuli appear to be processed within the same retinotopically-organized areas of cortex (e.g., Nishida, Sadaki, Murakami, Watanabe, & Tootell, 2003), yet some areas may have cells that prefer second-order stimuli (e.g., Dumoulin, Baker, Hess, & Evans, 2003). Findings of aging-related visual deficits that are more severe for second-order stimuli as compared to first-order stimuli have prompted the proposal (Faubert, 2002; Habak & Faubert, 2000) that older adults' visual abilities using second-order stimuli are more vulnerable because larger, more complex, and simultaneously engaged neural networks are required; in essence, second-order stimulus processing is more vulnerable than first-order processing because the computational load is increased. Further research is needed to evaluate the testable implications of this hypothesis for visual tasks and behaviors in everyday life. More recently, Tang and Zhou (2009) have pointed out, based on cross-sectional data, that aging-related deficits for second-order stimuli emerge earlier during the course of adulthood than do deficits to first-order stimuli, although the time course of decline is slower for first-order stimuli. A question that arises from this body of work is what sorts of visual tasks or behaviors (e.g., object recognition, face recognition, reading, route finding, driving) would be hampered at what point during adulthood given these reported selective vulnerabilities in visual processing, and what tasks are immune to aging effects. Furthermore, it is possible that the identification of such tasks, their characteristics, and their neural loci could help uncover the mechanistic underpinnings of visual processing deficits in the older brain, or at least set the field on a course to uncover these mechanisms. For example, Habak, Wilkinson, and Wilson (2008) showed that older adults have a processing speed similar to that of young adults when making same-view face discriminations, yet their processing speed is slowed when the task involves different views of the face. Their performance does not reach young adult levels even by increasing stimulus duration. The authors (Habak et al., 2008) point out that cortical activation during same-view face discrimination involves more extensive regions in older adults than young adults (Cabeza, Anderson, Locantore, & McIntosh, 2002; Grady, 2002; Grady, McIntosh, Horwitz, & Rapoport, 2000). They (Habak et al., 2008) argue, however, that the increase in cortical activation areas may not be sufficient for visually complex face tasks such as integrating face information across

multiple facial views, or, alternatively, older adults' broader cortical activation may commandeer networks that would normally have been used for more complex face processing.

Research has suggested that there is considerable neural adaptation in the aging visual system that supports the preservation, or maintenance, of certain aspects of visual perception during the adult life-course. As the discussion above implies, the field has largely been focused on how visual function is impaired as we grow older. But equally interesting is what aspects of visual processing and visual behavior are preserved and how does the brain "accomplish" this. Studying the instances of stability of visual function during aging could provide clues as to mechanisms of neural adaptation and plasticity in the central nervous system that mitigate the negative ramifications of aging-related structural and physiological changes in the brain. Examples of the preservation of function are thus quite interesting, and suggest that if the mechanisms supporting maintenance of function are identified, this potentially could inform the development of interventions to preserve visual functions that normally succumb to aging-associated degeneration. McIntosh et al. (1999) showed that both young and older adults were equally adept at a visual short-term memory task requiring the discrimination of two sine wave gratings. Yet positron emission tomography (PET) suggested that the neural systems used by young versus old adults were different in some respects. Occipital, temporal and inferior cortices and caudate were involved regardless of age. But for older adults, the functional interconnections between these regions were much weaker and the older adults recruited areas not used by younger adults to perform the task, namely medial temporal and dorsolateral prefrontal cortices (see also Bennett, Sekuler, McIntosh, & Della-Maggiore, 2001; Della-Maggiore, Sekuler, Grady, & Bennett, 2000).

Along similar lines, there are a number of examples in the vision and aging literature where a key aspect of visual perception remains constant throughout adulthood even though related aspects of the visual function are impaired – for example orientation tuning as estimated psychophysically, even though older adults require more contrast to discriminate the orientation of gratings (Delahunt, Hardy, & Werner, 2008). Other examples include the renormalization of chromatic mechanisms after cataract surgery (Delahunt, Webster, Ma, & Werner, 2004; Hardy, Frederick, Kay, & Werner, 2005), perceived contrast at suprathreshold levels (Beard, Yager, & Neufeld, 1994), and blur perception (Elliott, Hardy, Webster, & Werner, 2007; Jung & Kline, 2010) (see also Enoch, Werner, Haegerström-Portnoy, Lakshminarayanan, and Rynders (1999) and Werner (1996) for a discussion of these issues). These phenomena highlight a potentially remarkable tendency of the aging nervous system, at least under some circumstances, to reorganize or adapt itself to preserve visual perception and the visual behaviors it supports; these examples also highlight the possibility that deteriorating functions not spontaneously re-calibrated by the aging brain could be modified by perceptual learning through training or practice regimens. As discussed earlier, visual processing speed has already been shown to be amenable to improvement in older adults through practice (Ball et al., 2007; Yu et al., 2010), and thus, there may be reason for optimism that other aspects of visual processing can also be enhanced in older adults.

## 8. Conclusion

The motivation for research on vision and aging should spring from significance. Research on vision and aging over the next 25 years would best serve science and society if motivated by a theoretical framework or a practical need. It is not enough to describe and catalog all the various ways that older adults compares or fails to compare to younger adult's vision, and unfortunately

there have been some instances of this approach to research in the past 25 years. Of course there is always the possibility of a serendipitous discovery when engaged in description, but it can be argued that the most exciting and efficient scientific efforts will be those that programmatically build a basic evidence-base that also has the potential to address public health priorities. Significance in aging and vision research can assume a few different forms, and when we plan future scientific endeavors on this topic, it is useful to ask ourselves the following questions. First, how does the research contribute to our understanding of the fundamental processes involved in visual perception? Not just about aging per se, but theories and models about how we see. Just as human development is part of a comprehensive understanding of a visual phenomenon, so is aging. Second, is the research directed at a fundamental visual process that is critical in performing a task or behavior important in everyday life? Here it is important to keep in mind that just because one can demonstrate an aging-related deficit or difference in some task in the psychophysics laboratory, this does not necessarily mean that this deficit is relevant to understanding older adults' everyday visual performance or behavior difficulties. Whether a deficit negatively impacts everyday function and well-being is actually an empirical question in that everyday ramifications of the deficit must be established. Third, does an aging-associated deficit, particularly if it is accentuated in some individuals, serve as an advance "warning sign" of a degenerative condition of aging (e.g., AMD, glaucomatous optic neuropathy)? How could knowledge of this putative signal and its underlying causes be exploited to help understand the pathogenesis of the condition and ultimately to prevent or arrest the development of the disease? By this third point, we are not arguing that all aging and vision research has to have clinical significance. Yet researchers in aging and vision should always be mindful that degenerative conditions of the eye and brain are prevalent in later adulthood, and discoveries in aging and vision process can be relevant to these pathogenetic processes.

In general, there is an under-appreciation of individual differences in the aging of visual function. The most popular study design in the field to date involves the comparison of young versus old group differences. While there are undoubtedly "general principles" in the aging of visual function (some of which are discussed in this article), older adults are individuals with varying lifestyle, genetic, and environmental exposures during the life-course that can theoretically impact ocular and brain structure and function in later life in different ways. For this reason, there is little theoretical basis for assuming that a visual processing deficit demonstrated in a handful of older adults is in fact universal. Differences in genetic, environmental and lifestyle factors in older adults could increase or reduce the risk of certain types of aging-associated visual deficits, an issue that has received scant attention in previous research. By methodological necessity such studies typically require large sample sizes, yet some of the most exciting discoveries in vision and aging research from an etiologic perspective may be those that identify the characteristics of those who visually age well versus those who exhibit more serious visual losses that interfere with visual performance and quality of life. Once those markers, causes, and/or risks are identified, research can focus on using this information to develop interventions to prevent, reduce, or side-step their impact, the ultimate goal being to preserve a high-level of visual function into the advanced years of adulthood.

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