Table 1. Amino acid composition of hemerythrin.

Amino acid	Residues per 13,500 g of protein
Lysine	10.5
Histidine	6.5
Arginine	3.03
Aspartic acid	16.2
Threonine	4.35
Serine	3.38
Glutamic acid	9.5
Proline	4.03
Glycine	6.2
Alamine	5.3
½ Cystine	0.96
Valine	3.72
Methionine	0.85
Isoleucine	8.34
Leucine	7.36
Tyrosine	4.8
Phenylalanine	8.5
Ammonia	12.2

alike one would expect 14 to 15 peptides in a peptide map of a tryptic hydrolysate, whereas the number actually found is 28 to 30. The same number of tryptic peptides has been reported by Manwell (*4*).

The most obvious interpretation of these results is that the subunits of hemerythrin are of two distinct types with major differences in the respective sequences of amino acids. On the other hand, peptide maps of chymotryptic hydrolysates of either pooled hemerythrin or protein from individual worms do not show substantially more peptide spots than can be accounted for by the sum of the number of amino acid residues providing peptide bonds known to be susceptible to cleavage by this enzyme. All attempts to separate the presumed different subunits have failed. These attempts included (i) chromatography on DEAE-Sephadex, CM-Sephadex, and Amberlite IRC-50 under a variety of conditions, with and without urea, and (ii) electrophoresis in starch gel under different conditions and with a variety of dissociating reagents. Some of these efforts have produced resolvable peaks but these peaks have shown peptide maps identical with those from the original protein. An alternative interpretation must therefore be borne in mind, namely, that the subunits are basically identical (*4*) but that the linkages susceptible to attack by trypsin are so grouped that more peptides are produced than would be expected from the number of susceptible bonds, because of occasional partial hydrolysis.

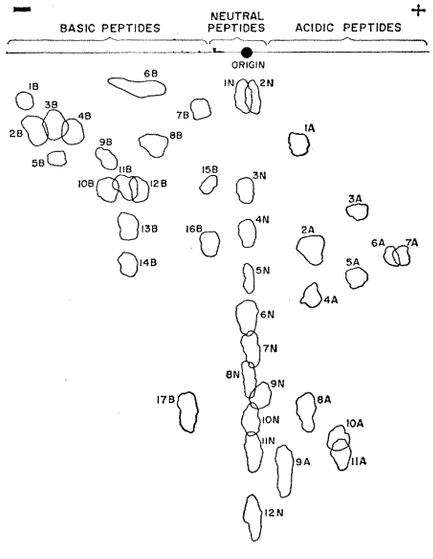

Fig. 1. Peptide map of a chymotryptic hydrolysate of hemerythrin.

A further complication in the structure of hemerythrin arises from observations of differences in peptide maps of enzymic digests of the protein of individual animals. One such difference has been reported by Manwell (*4*). We have found in the chymotryptic digests, differences indicative of substitutions of amino acids in at least one, and possibly more, positions in the chain. Figure 1 shows the peptide map of the chymotryptic digest of pooled hemerythrin. When similar chymotryptic digests of the hemerythrin of individual worms are examined, a class of worms may be distinguished, in which the peptides marked 13B (basic, tryptophan-containing) and 3A (acidic, tyrosine-containing) are present, and another in which they are absent. These two peptides are invariably either present or absent together. This suggests the substitution of a residue by either tyrosine or tryptophan, thus affording a new point of chymotryptic attack, or the deletion of a portion of the peptide chain.

The variation in peptides 13B and 3A is the most obvious because they reveal amino acids which are easily detected by specific color reagents. It is probably not the only one that exists in hemerythrins from the species *Golfingia gouldii*. Manwell (*4*) has found variations in electrophoretic behavior in the protein from individual worms of the same species which he suggests may be due to a single substitution of an acidic residue for an uncharged one.

Thus while the results of tryptic hydrolyses indicate the presence of two kinds of subunits, this conclusion is not unequivocal since the observations from chymotryptic hydrolyses could also be rationalized in terms of a single chain. In either event it is clear that variations in peptide constitution of the subunits do occur from one individual to another (*8*).

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Adaptation to Chromatic Aberration by the Human Visual System

Abstract. *Prolonged exposure to the chromatic dispersion produced by prism spectacles leads to a perceptual adaptation. The adaptation develops rapidly in the first two days of the wearing of the spectacles, and seems to be a special reaction to the relative change in illuminance at intensity gradients on the retina.*

Perceptual adaptation to changes of the eyes' normal optical linkage to the environment may be experienced by anyone who, wearing a new pair of glasses, is at first disturbed by the distortional side-effects. Such adaptation suggests the existence of regulating mechanisms underlying normal vision, and provides a novel perspective on the dependence of perception on past experience. Among the most interesting of these adaptation phenomena are those reported by Gibson (*1*) and Kohler (*2*) for the image aberrations

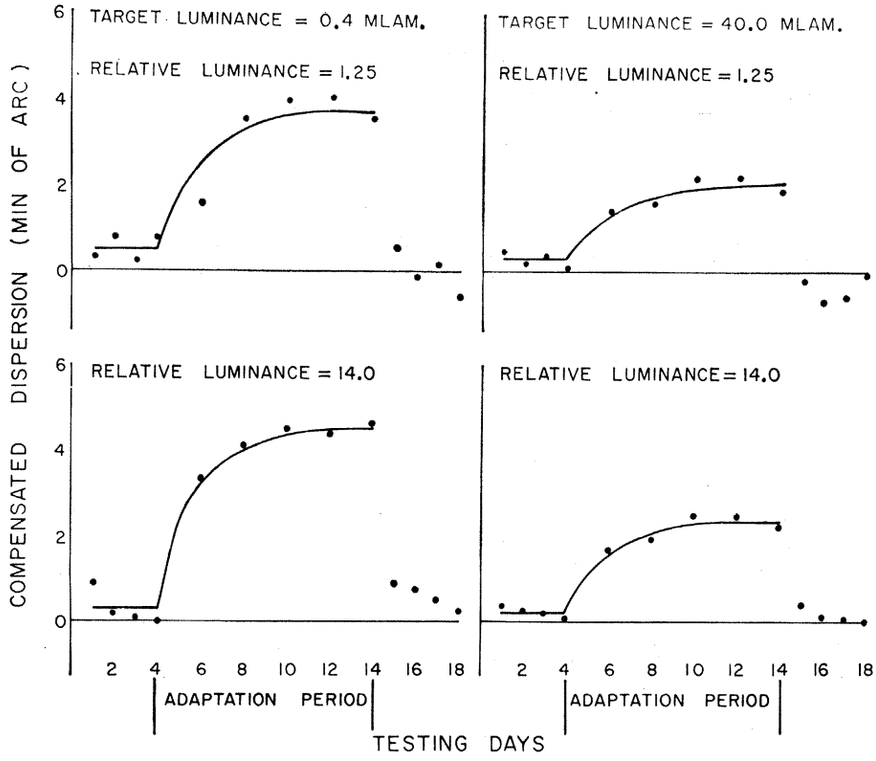

Fig. 1. Development of compensation for chromatic dispersion during prism spectacle wearing. The ordinate values are the mean amounts of prismatic dispersion present when the subjects reported no visual color fringes on the target. The data are shown for four conditions of absolute and relative target luminance.

produced by prism spectacles. In contrast to the rapid correction of prism-induced visual-motor errors (*3*), these slower developing corrections of the visual appearance of the environment seem to involve alterations of basic sensory functions.

We have investigated adaptation to the chromatic dispersion of the retinal image produced by prism spectacles, extending Kohler's self-observations of this phenomenon. When prisms are mounted with identical orientations before the two eyes, the dispersion they produce manifests itself visually in the appearance of color fringes wherever the short- and long-wavelength components of the retinal image fail to overlap. With base-left prisms, for example, bluish fringes are seen on the right of light regions, and reddish fringes on the left. After prolonged wearing, the strength of the fringes may noticeably diminish. More striking, an opposition illusion takes place after the spectacles are removed: blue fringes on the left of light regions, and red on the right.

Kohler made three important observations on these after-effect color fringes as he experienced them: (i) They behaved like the original prismatic fringes in remaining tied to the edges of objects during eye movement. In this they were different from ordinary negative after-images of color, which move with the eyes. (ii) They appeared both in polychromatic and monochromatic illumination, but only if the object differed in brightness from its background. In this they were unlike the prismatic fringes. (iii) They were more definite in weak illumination than in strong, again unlike the prismatic ones.

Observations (i) and (ii) suggest that the adaptation to prismatic dispersion consists of a special reaction of the visual system to intensity gradients in the retinal image, gradients of a kind related to those which ordinarily elicit simple edge perception. This adaptation response manifests itself in an attenuation of prism-produced color fringes and, in the absence of prismatic dispersion, in the appearance of reversed color fringes. Observation (iii) seems to be a clue to just what properties of an intensity gradient evoke the adaptation response.

We have measured the development of this adaptation response in six subjects, and tested its dependence on various parameters of image intensity gradient. Our subjects, who had good uncorrected vision, wore 20-prism diopter spectacles continuously for 10 days, removing them only for the daily battery of tests that lasted an hour.

The adaptation response was measured by an indirect technique suggested by Kohler; its power to compensate for prismatic dispersion was determined. With the prism spectacles removed, the subject viewed an achromatic test pattern of vertical stripes through a Hughes variable prism. His task was to eliminate any apparent color fringes by adjusting the power of the variable prism. These "null-adjustments" should be close to zero-prism diopters before wearing prism spectacles, evincing the subject's sensitivity to prismatic dispersion. With the development of an adaptation response during spectacle wearing, the null-adjustments should change in the direction of the spectacle prism power. The amount of prismatic dispersion present when the subject sees no color fringes is our measure of the dispersion-compensation power of the adaptation response.

This test was carried out under two kinds of illumination: (i) narrow-band illumination with a peak at 642 mμ and a half-width of 15 mμ; and (ii) full-spectrum illumination produced by a tungsten lamp with a color temperature of 3200°K.

With the narrow-band illumination, the strong saturation of the illuminant hue seemed to obscure color fringes. However, three of our subjects consistently reported fringes of blue and an intense red after ten days of spectacle wearing. Confirmatory evidence that these color fringes were in fact produced within the visual system, and not by some residual dispersion of the retinal image, was provided by the fact that adjusting the variable prism had no effect on them.

With full-spectrum illumination, subjects could always find a setting of the variable prism which eliminated color fringes. On either side of this setting, fringes of opposite hue would be produced. At this "visual null point" the adaptation response must have exactly compensated for the image dispersion present.

Figure 1 illustrates the development of this compensation for prismatic dispersion, evincing the growth of an adaptation response. The subjects were tested for 4 days prior to putting on the spectacles; then on alternate days during the wearing period. Three of

the subjects were tested for 4 days after removal; the others continued wearing the spectacles, in connection with a study of adaptation to spatial distortion. Prismatic dispersion is indicated in terms of a standard measure, namely, the relative angular displacement of the Fraunhofer C and F lines, which bracket the region of the spectrum to which the eyes are most sensitive. In terms of this measure, the spectacles worn by the subjects had a dispersion of 10.2 minutes of arc. The data points show the mean compensation power of the subjects' eyes, that is, the amount of prismatic dispersion in the variable prism which was just balanced by the adaptation response. The solid lines describe the data in terms of a steady state before spectacle-wearing and an exponential growth process during wearing.

Two levels each of target-stripe absolute luminance (L_1) and luminance relative to background (L_1/L_2) were tested, giving the four developmental curves shown in Fig. 1. Changing the relative luminance from 1.25 to 14.0 produced no important change in the eyes' compensation power. Changing the absolute luminance produced a change in compensation power which is statistically significant ($p \leq .01$); a hundred-fold increase in absolute target luminance reduced the compensation power by approximately one-half.

The same order of inverse relationship between compensation power and absolute target luminance was more general, in a special test series on the tenth day, throughout a luminance range from 10^{-3} to 40 mlam. At the lower end of this range, the compensation power actually exceeded the dispersion power of the spectacles. Below this region of ordinary color sensitivity, no color fringes were seen. Therefore although polychromatic light is not necessary to evoke the adaptation response, stimulation of the receptors participating in wavelength discrimination is necessary.

The effects of relative and absolute target luminance on the adaptation response itself cannot be directly inferred from their effects on its power to compensate for prismatic dispersion. Indeed, an analysis of the joint effects of prismatic dispersion and target luminance on the retinal image indicates that the adaptation response must vary with relative target luminance, and to a lesser degree—if at all—with absolute target luminance. Our reasoning is as follows.

The angular dispersion of the prism, which we measured to determine compensation power, produces a relative displacement of the long- and short-wavelength components of the target image. Thus the long-wavelength image of the target is partially superimposed on the short-wavelength image of the background, and vice versa. The color fringes which a prism initially produces are due, then, to the difference between the luminance of the target and that of the background. This luminance difference will be increased both by increasing absolute target luminance alone (by increasing target and background luminance in proportion), and by increasing relative target luminance alone (by decreasing background luminance). In accordance with this, the prism-produced color fringes are more vivid in high illumination than in low, and along high-contrast borders than along low-contrast borders.

This means that for a fixed amount of angular dispersion, a constant level of compensation would require that adaptation response strength increase with increases of either absolute or relative target luminance. Since we found that the amount of compensated dispersion did not change significantly with increases of relative target luminance, it follows that the strength of the adaptation response must have increased in proportion to relative target luminance. On the other hand, since the amount of compensated dispersion decreased with increases of absolute target luminance, we infer that the adaptation response does not change in proportion to absolute target luminance.

We may hypothesize, therefore, that the adaptation response underlying compensation for prismatic dispersion consists of an alteration of some normal psychophysical function of relative target luminance, such as contour formation or color contrast.

The possibility of similar adaptation to the chromatic aberration produced by spherical spectacle lenses, or to the intrinsic chromatic aberration of the eye (*4*), is not established. The dispersion produced by these differs in certain important respects from that of the prism spectacles. As a fundamental test, however, it may be suggested that wherever adaptation such as that we have studied takes place, it should manifest itself in ineradicable color fringes along high contrast borders in monochromatic illumination.

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Behavioral Response Rates in Pigeons: Effect of α-Methyl-m-tyrosine

Abstract. *An intramuscular injection of α-methyl-*m*-tyrosine (100 mg/kg), which differentially depletes serotonin and norepinephrine in both brain and heart, was given to two groups of pigeons trained to peck at a key for food. The first group received an injection 12 hours before the daily session and showed no behavioral effect. Response rates of birds in the second group, which were injected 30 minutes after the start of the daily session, decreased and returned to normal within 9 hours after injection. Preliminary data on brain serotonin of pigeons indicate that the disruption of the behavior follows the same time course as the change in serotonin.*

Since 1954, data have been accumulated which suggest that 5-hydroxytryptamine (serotonin) has an important function in the central nervous system and that variations in the physiological levels of this amine affect behavior. One way to investigate the effect of serotonin upon the central nervous system is to correlate the abnormal levels of this amine with changes in behavior. In-