

Failure of intercellular adhesion in hair fibers with regard to hair condition and strain conditions

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Synopsis

Although adhesion failure in hair fibers can occur inside cells, it occurs more frequently in the cell membrane complex (CMC), often involving the rupture of interlayer bonds. Therefore, a model of the CMC is presented, based on prior research in which we propose interconnecting bonds between the layers to assist in our interpretation of hair-fracturing mechanisms for cuticle chipping, deep transverse cuticle cracks, cracks during heat drying, scale lifting by surfactants, and catastrophic failure. Failure in the wet state generally involves hydrophilic layers, e.g., the contact zone of the CMC or the endocuticle or bonding to these hydrophilic layers, whereas failure in the dry state generally involves bonding between hydrophobic layers, e.g., beta-delta failure. Chemical damage by perms, bleaches, and sunlight, by breaking specific chemical bonds, influences the sites of initial failure and increases the number of routes for crack propagation, leading to more complex fracture patterns.

INTRODUCTION

Failure of adhesion can occur at different sites in hair fibers, depending on the condition of the hair and the conditions of strain that produce failure. The condition of the hair is determined primarily by prior treatments and fiber twists and/or flaws, including whether or not it contains a medulla and whether it has been chemically treated or exposed to sunlight, and the kind and amount of sorbed materials. The conditions of strain include stretching, bending, twisting, percentage deformation, the employment of heat such as heat drying, and water content or percent relative humidity.

The cosmetic importance of adhesion failure resides in the fact that it controls hair breakage and fragmentation, actions that are so important to the look, the feel, and the condition of the hair. Fragmentation of hair is determined primarily by chemical treatments to the fibers, including sunlight exposure, the conditions of deformation, and abrasive actions that are employed in everyday grooming actions (1). It is the combination of these actions at high water content that results in the most severe hair erosion, especially when combined with repetitive actions, as in extension cycling, fatiguing, and abrasion to break.

Although failure can occur inside cuticle and cortical cells, the cell membrane complex (CMC) is involved in several different types of adhesion failure and frequently is the origin of fracture and crack propagation. Therefore, a model of the CMC is provided in this paper to help explain where and how failure occurs under several different conditions.

HAIR FIBER STRUCTURE

The CMC is an interconnecting unit consisting of three different types that are very similar structurally (see Figure 1). The CMC between cuticle cells is called cuticle-cuticle CMC, at the junction of the cuticle and the cortex is the cuticle-cortex CMC, and the CMC between cortical cells is called cortex-cortex CMC. Of these three structures the cuticle-cuticle CMC and the cortex-cortex CMC have been studied to the greatest extent. The subtle known differences in these structures are that the cuticle-cuticle CMC contains 18-methyl eicosanoic acid (18-MEA) in one of its beta layers (outer beta layer, the one immediately on top of cuticle cells), while the other two CMCs do not contain this specific fatty acid, but consist of straight-chained fatty acids from C12 through C18 and oleic acid in place of this unique branched fatty acid (see Figure 2A). 18-MEA is distinctive in its role in the cuticle-cuticle CMC (2), which will be explained later in this paper. Since 18-MEA is a C21 fatty acid and exists only in the outer beta layer of the cuticle-cuticle CMC (3) and is replaced by shorter-chain fatty acids in the cortex-cortex CMC, the cuticle-cuticle CMC should be thicker than the cortex-cortex CMC. The cuticle-cuticle CMC has been shown to link fatty acid (18-MEA) to cuticle cell membranes in the outer beta layer (Figure 2A) through thioester linkages (4), while it would appear from the work of Bryson *et al.* (5) that fatty acids in the cortex-cortex CMC are linked to the hydrophobic keratin protein layer through ester linkages.

THE STRUCTURE OF THE CUTICLE-CUTICLE CMC

Nearly three decades ago, Fraser and coworkers (6) described the CMC as consisting of

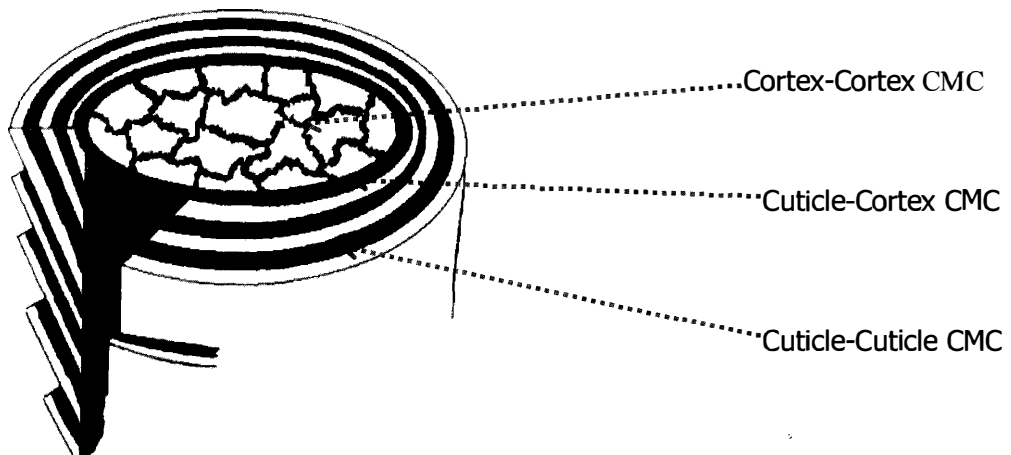


Figure 1. The CMC, a structure where failure tends to occur. There are three types of CMC in human hair that are structurally different.

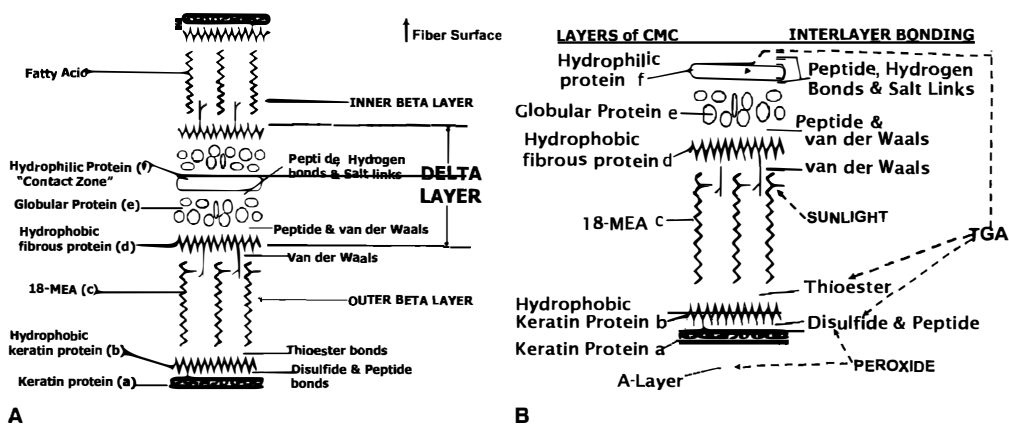


Figure 2. A. The monolayer model for the cuticle-cuticle CMC, with principle bonding proposed between layers. The inner beta layer is the "lower" and the outer is the "upper" beta layer. B. The monolayer model for the cuticle-cuticle CMC (lower half) and its principle interlayer bonds, with some primary sites for chemical attack.

two lipid layers (called inert beta layers) separated by intercellular cement, the delta layer (Figure 2A). Several important developments have occurred since Fraser's description of the CMC that have further improved our understanding of this important structure. Among these important developments are the work of Rivett and coworkers (3,4) and Bryson and coworkers (5). Two different models are presented in this paper in which this prior research is taken into consideration to provide a more detailed chemical representation of the CMC. In our models, we are proposing the interconnecting bonds between the different layers of the CMC to assist in our interpretation of hair-fracturing mechanisms. Both of our models consist of protein and fatty acid layers. Model 1 (Figure 2A,B) contains monolayers in the beta layers, while model 2 (Figure 3) contains bilayers in the fatty acid domains consistent with Fraser's original concept.

We are also suggesting a change in terminology. Up to this point, the beta layer immediately on top of each cuticle cell has been called the upper beta layer and the one beneath each cuticle cell has been called the lower beta layer. This terminology is confusing, especially for schematics of the CMC as in Figures 2A,B and 3. Therefore, we propose that the beta layer that sits on top of each cuticle cell and often becomes the outer hair surface be called the outer beta layer and the one immediately beneath each cuticle cell be called the inner beta layer (see Figures 2A,B and 3), and this terminology will be used throughout this manuscript.

Figure 2B shows only one-half of the CMC structure for the monolayer model and depicts some of the primary sites for chemical attack by permanent waves, bleaches, and sunlight. The top half of the CMC is not depicted in this figure because it is similar to the lower half, with the exception that it does not contain 18-MEA but contains saturated fatty acids (C12-C18) and oleic acid in place of 18-MEA. Our preference is for the monolayer model over the bilayer model for the following reasons:

- Swift (2) has pointed out that a monolayer model fits better from the point of view of CMC measurements.
- If the beta layers are monolayers, then 18-MEA is linked to the delta layer through short hydrophobic side chains rather than through ester or thioester bonds (in the

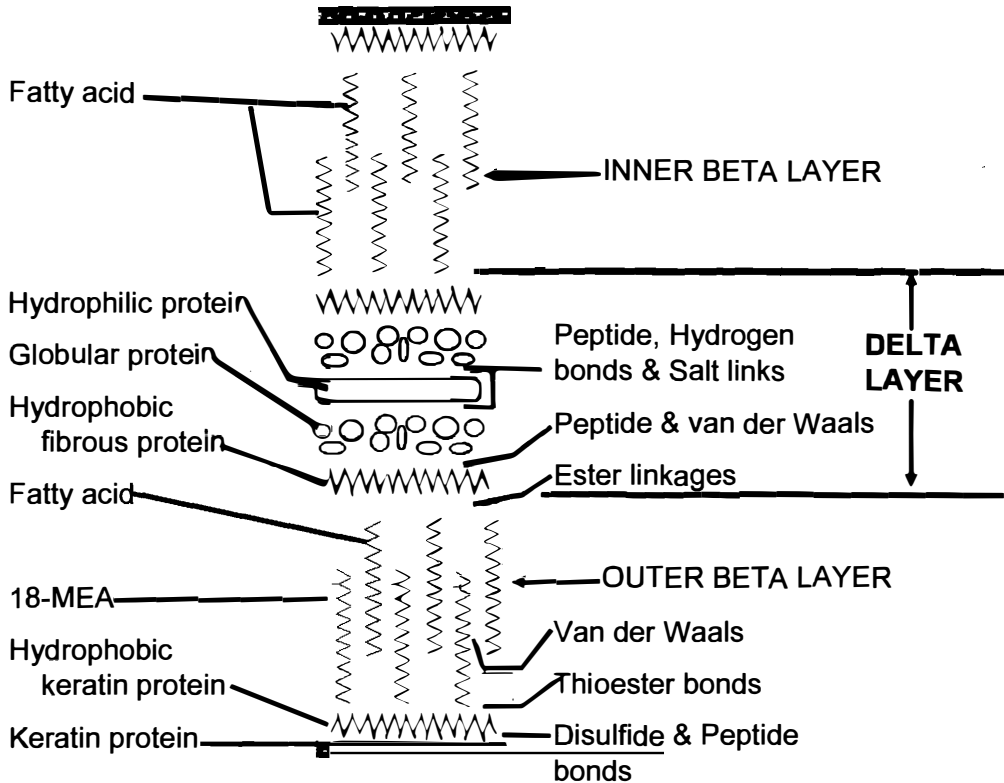


Figure 3. The bilayer model for the cuticle-cuticle CMC, with principle bonding proposed between layers.

bilayer model). Some of these hydrophobic amino-acid side chains in the monolayer model are also branched, making the outer beta layer even more susceptible to failure at the delta layer, where it has been shown to occur (7).

- The monolayer model contains fewer layers of ester/thioester linkages; thus it will be more specific in its reactions to nucleophilic agents like mercaptans, hydroxide, and hydroperoxide anions—a point addressed later in this paper.

Another point of contention concerning the CMC is whether or not the delta layer contains globular proteins or glycoproteins (2). We believe the current evidence favors globular protein layers in the delta layer for the following reasons:

- No further evidence for glycoprotein in the CMC has been presented since 1991 (8); however, Bryson *et al.* in 1995 (5) isolated lipid-soluble lipoproteins from the delta layer of cortex-cortex CMC and not glycoproteins, favoring the globular protein model.
- The delta layer resists solubilization by reducing or oxidizing agents and by acids and alkalis (5). If the CMC contains globular proteins similar to those in many other membranes containing large domains of hydrophobic amino acids on their surfaces (9), then these should be resistant to aqueous reagents as Bryson found. Thus the globular protein model is consistent with its known reactivity.
- The delta layer swells only about 10–15% (10); therefore, much of it is hydrophobic. Such a relatively hydrophobic delta layer is more consistent with a globular protein model than with a more hydrophilic glycoprotein.

- The delta layer stains with PTA. This is either a reaction of hydroxyl groups of a polysaccharide or of a primary amine function. Swift (2) has explained that this reaction is blocked with FDNB; therefore, it is more likely a reaction involving primary amine groups, consistent with a globular protein.
- The delta layer reacts with periodic acid/silver methenamine (2), a method for polysaccharides; however, since cystine interferes with this reaction, it is still consistent with a globular protein in the delta layer.

We believe that these models, with their proposed interconnecting bonds, help to explain where failure can occur and how it occurs, and we will use these models in that manner in the following discussion.

SOME GENERAL RULES OF ADHESION FAILURE

Strain (the amount of deformation) more than stress determines how failure occurs in hair fibers. Stress-strain curves in the wet versus the dry state show much greater strain (deformation) in the wet state prior to entering into each successive region of the curve or even breakage (11). Faster rates of extension tend to inhibit the diversion of cracks in the axial direction and thus to promote smooth fractures (12). This is why fibers that can be extended to very high strains (particularly in the wet state) and are in good condition, i.e., undamaged with no flaws, tend to fracture rapidly at high strains, providing smooth fractures (Figure 4A).

Undamaged hair with no flaws, including no medulla when extended to break, is more likely to provide smooth fractures under most conditions, but especially at high humidities or when wet. Most Caucasian hair fibers in good condition and relatively straight with no twists, when extended to break in water or at very high humidities, provide mostly smooth fractures with crack initiation near the periphery of the fiber, often at the cuticle-cortex CMC. At lower humidities, extension provides mainly step fractures (see Figure 5), although under very dry conditions, smooth fractures are also observed with crack initiation in the cortex (Figure 4B).

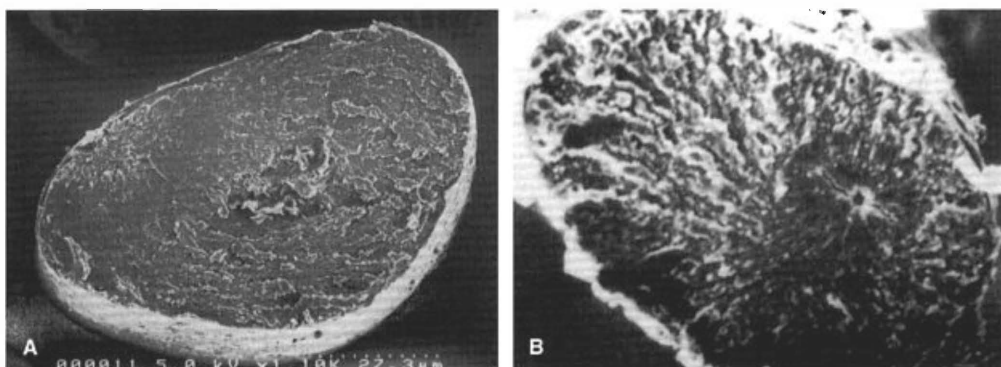


Figure 4. A. Extension to break when wet generally gives smooth fractures, high RH (100%). SEM by S. Ruetsch, TRI Princeton. B. Extension to break when very dry can give smooth fractures (12). Reprinted with permission of the *Journal of Applied Polymer Science*.

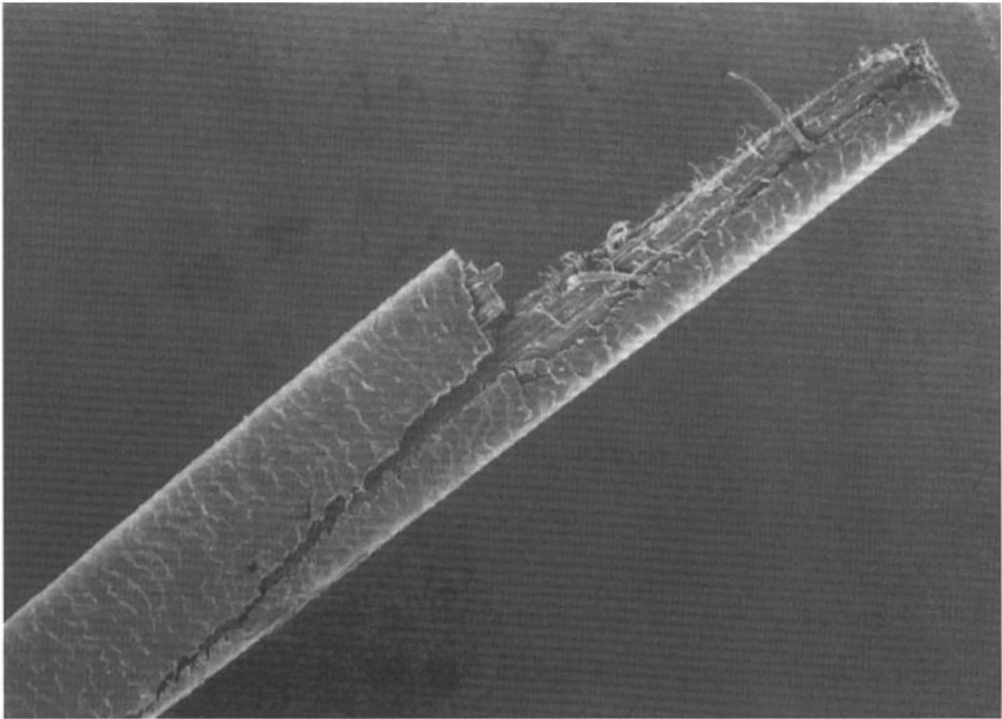


Figure 5. Extension to break when dry generally gives step fractures (S. Ruetsch, private communication). SEM by S. Ruetsch, TRI Princeton.

Crack initiation is generally along the cross section, while more complicated fractures occur by crack propagation in the axial direction. For example, fibers with flaws (including a medulla) or with twists, or chemically damaged fibers, provide more complicated fractures, such as multiple-step fractures, angle fractures (a type of step fracture), fibrillation, or splits. These more complicated fractures arise through axial propagation of cracks through the CMC, or through flaws (including a medulla) or damaged areas, especially the CMC.

DRY-STATE OR LOW-HUMIDITY FAILURE

Deformation in the dry state (at low relative humidity) first produces scale edge lifting, and then with continued deformation, catastrophic failure occurs.

BETA-DELTA VERSUS BETA-BETA FAILURE

Extension cycling or extension at lower strain rates, at low RH, causes scale lifting and buckling of individual scales (see Figures 6, 7) rather than deep transverse cracks in the cuticle as occurs at high relative humidities or when wet at high strain rates. The extent of scale lifting and buckling of scales depends on the percentage extension and/or the number of cycles extended (12,18).

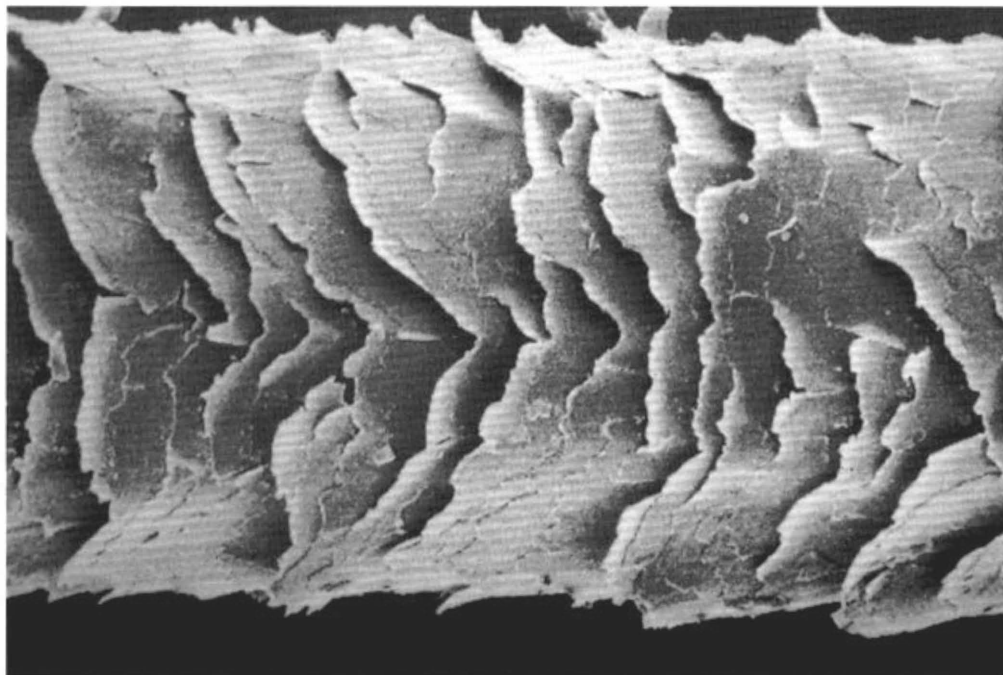


Figure 6. Scale lifting and buckling from extension (S. Ruetsch, private communication). SEM by S. Ruetsch, TRI Princeton.

Failure of adhesion between overlapping scales in the cuticle is thought to occur at the hydrophobic outer beta layer, between the outer beta layer and the delta layer as first described by Negri *et al.* (7). Failure at this site occurs because of the weak hydrophobic bonding between branched hydrocarbons (18-MEA) and relatively short hydrocarbon amino-acid side chains of the hydrophobic fibrous protein layer (d), as depicted in the monolayer model of Figure 2A. This site is conducive to failure when the cuticle layers are strained at low moisture levels and allows the flow of cuticle past cuticle during fiber extension (2,12,14), during extension cycling (13), or even during bending (13,15), particularly in the dry state at relative humidities of 65% or lower. The lower the RH (the moisture content of the hair) the lower the strain at which failure of the scale structure can be initiated between the outer beta layer and the delta layer (13).

If the bilayer model (Figure 3) exists, then this type of scale lifting failure would occur between the two fatty-acid bilayers, i.e., between the 18-MEA layer and the fatty acid layer of Figure 3, producing beta-beta failure. This type of failure is not consistent with the conclusions of Negri *et al.* (7), providing support for the monolayer model over the bilayer model.

The different layers of the hair structure are interconnected and stretch dependently. In the dry state, failure often occurs in or between hydrophobic layers because hydrophilic layers are not as extensible at lower moisture levels and therefore force higher strains in the hydrophobic layers for any given stress.

FIBER FRACTURE AT LOW RH

Extension to break, at lower RH (primarily lower than 30–45% RH), of chemically

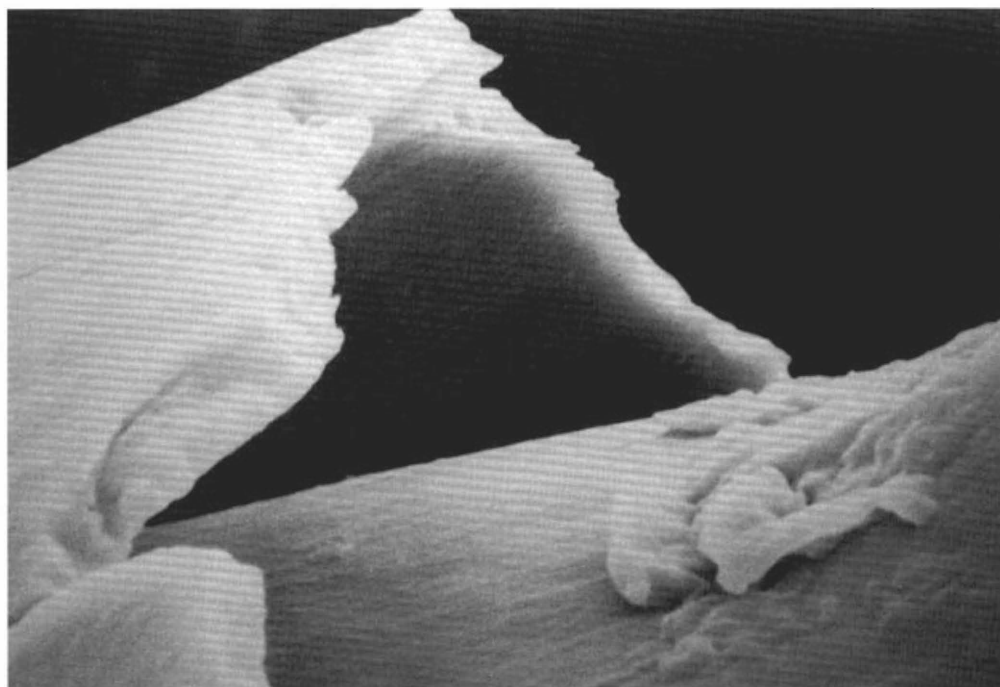


Figure 7. Beta-delta failure from extension of hair fibers in the dry state (21). Reprinted with permission of the *Journal of Cosmetic Science*.

unaltered hair generally provides step fractures rather than smooth fractures. Under this condition, crack initiation is often in the cortex and is initiated at lower strain levels than in the wet state. Therefore, crack propagation occurs at a slower rate. Flaws such as the presence of the medulla (more frequent in thicker fibers) and the intercellular cement can play a role in directing crack propagation in the axial direction to provide step fractures (12). At very low humidity, smooth radial fractures are also observed, with crack initiation in the cortex and brittle failure along the fiber cross section (Figure 4B).

CUTICLE CHIPPING VIA EDGE FAILURE IN THE DRY STATE

Chipping away of the cuticle edges is a common occurrence in everyday grooming actions (Figure 8). In the dry state, edge failure most likely begins as outer beta-delta failure, between 18-MEA and the delta layer in the monolayer model (as described), because of weak hydrophobic bonding (Figure 2A), followed by scale edge lifting (Figure 7). Shearing off of scale fragments then results from rubbing actions, as occurs in combing, brushing, or even shampooing.

WET-STATE OR HIGH-HUMIDITY FAILURE

Deformation in the wet state first leads to deep transverse cracks across the cuticle, and then with continued deformation fiber breakage occurs.

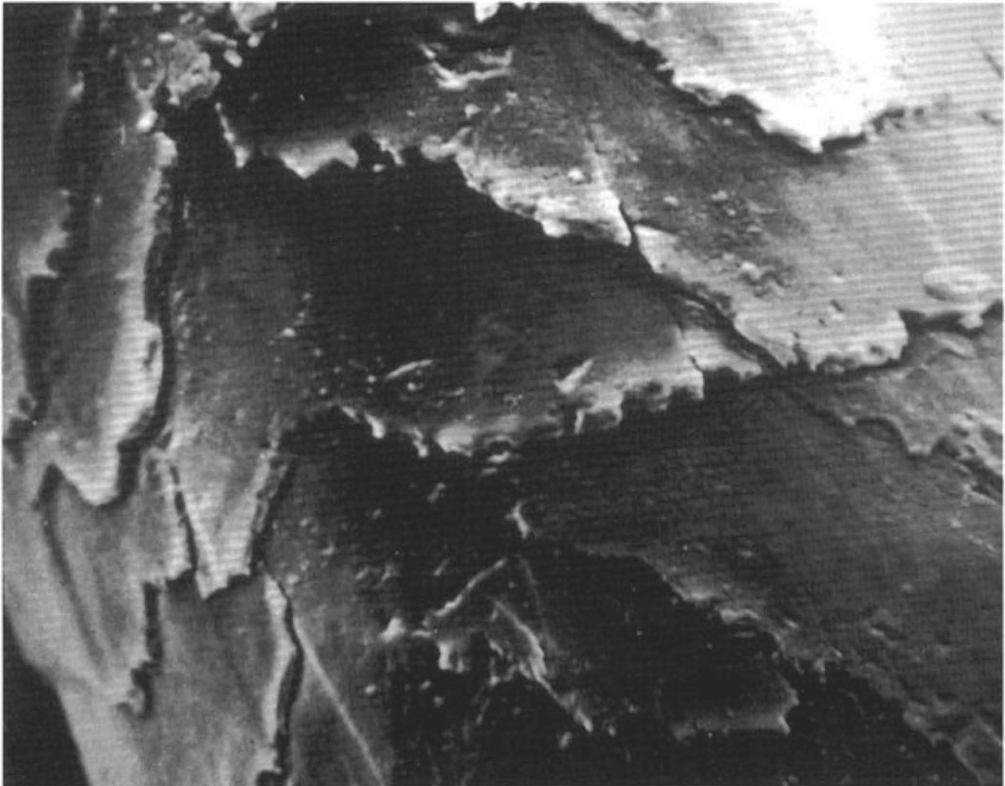


Figure 8. Cuticle chipping from dry combing hair fibers.

DEEP TRANSVERSE CRACKS ACROSS THE CUTICLE

Hydrophilic regions are swollen more at high humidity or in water and are more deformable than in the dry state. The swollen cortex exerts pressure on the cuticle-cortex CMC (Figure 1), thus we conclude that the hydrophilic “contact zone” (Figures 2A, 3) of the delta layer between the cortex and the cuticle becomes the weak link from Poisson contraction and swelling pressure, as suggested earlier (12). Therefore, crack initiation occurs in the cuticle-cortex CMC very likely near the interface of the contact zone (hydrophilic protein layer) and the globular protein layer (see the monolayer model of Figure 2A). Under the influence of swelling pressure, crack propagation during deformation extends across all cuticle layers perpendicular to the fiber axis to produce deep transverse cracks (Figure 9), and virtually no scale lifting or buckling occurs if the fibers are not previously damaged (13).

EXTENSION TO BREAK OR CATASTROPHIC FAILURE

Continued extension or deformation leads to fiber breakage, with high moisture levels providing mainly smooth fractures for Caucasian hair, especially hair with little to no damage (see Figure 4A) (12). In the wet state, especially when the hair is not damaged or does not contain flaws, hydrophilic layers near hydrophobic layers are swollen and more extensible than in the dry state and, to some extent, relieve strain in the hydro-

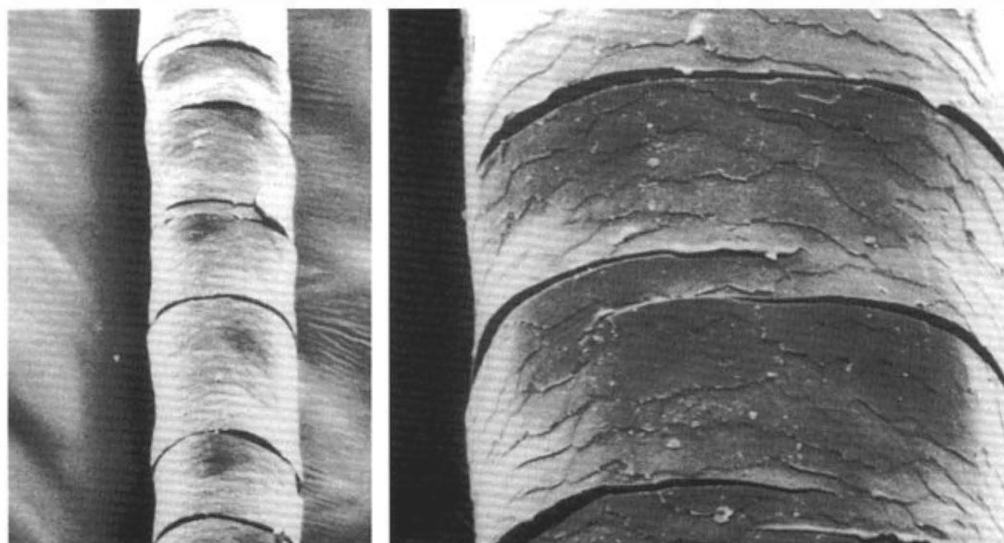


Figure 9. Deep cuticle cracks perpendicular to the fiber axis from extension cycling at high RH (13). Reprinted with permission of the *Journal of the Society of Cosmetic Chemists*.

phobic layers because of their greater extensibility. Thus, greater strains are allowed for any stress (at high moisture levels), and all layers are stretched close to their limit so that when failure occurs, crack propagation can be rapid and “uniform” through all layers, providing smooth fractures.

At high RH, crack initiation is often near the periphery, in the proximity of or in the cuticle-cortex CMC (Figure 1), and when wet, cortical cells are weakened and strained to their limit; thus failure can occur within the cells themselves. As discussed by Kamath and Weigmann (12), the swelling pressure on the cortex, in conjunction with Poisson contraction and tensile forces acting along the fiber axis, creates intense pressure on the cortical cells near the cuticle-cortex junction, leading to crack initiation that propagates rapidly toward the center of the cortex and provides a smooth fracture.

Negroid hair fibers behave differently because of their natural twists and flaws caused by damage from grooming and product treatments. This type of hair, even when wet, tends to give step fractures (42%) (with much longer lengths of axial fracture), fibrillation (24%), and angle fractures (21%), as compared to Caucasian hair with 91% smooth fractures and only 9% step fractures (16).

FAILURE OF ADHESION DURING HEAT DRYING OF HUMAN HAIR

Gamez-Garcia (17) has shown that during heat drying, because of evaporation of water from the hair surface, the top layers of the uppermost cuticle cells become more rigid and brittle than the underlying layers and a weakness first appears as white lines on the cuticle surface. These lines run parallel to the axis of the fiber. On further wetting and blow-drying, cuticle cracks become fully developed through the surface and top layers of the uppermost scales. In many SEMs the cracks can be seen to extend laterally beneath the surface in the uppermost scale layer (Figure 10), most likely at or in the endocu-

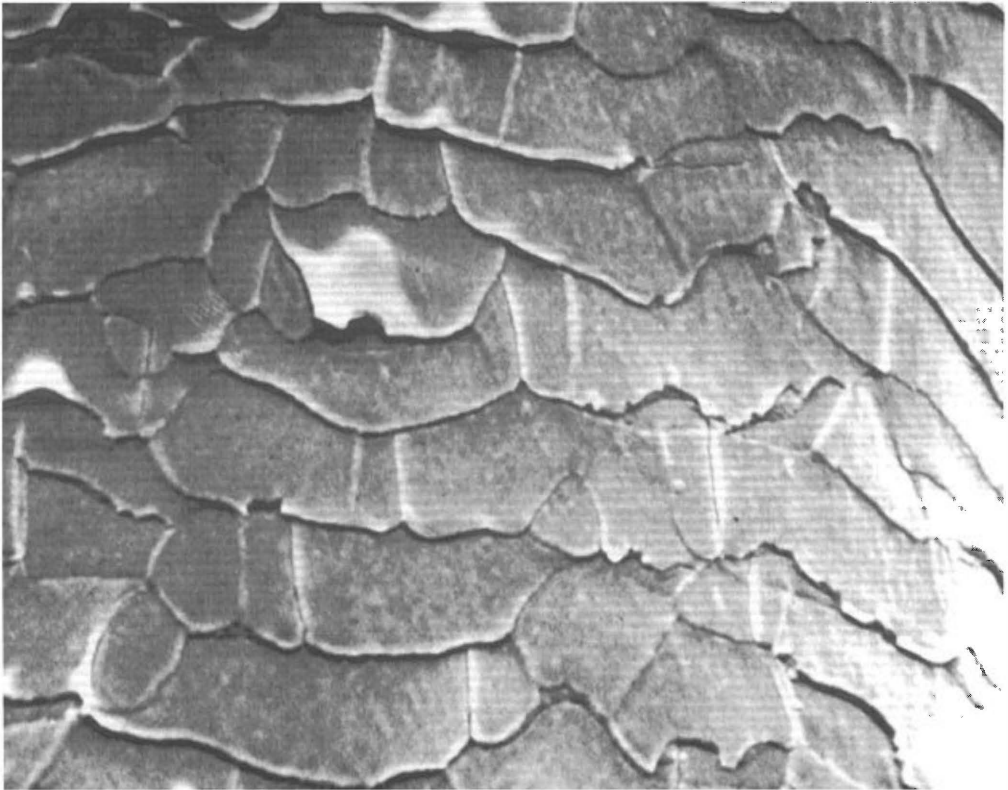


Figure 10. Cracks parallel to the fiber axis from heat drying hair (17). Reprinted with permission of the *Journal of the Society of Cosmetic Chemists*.

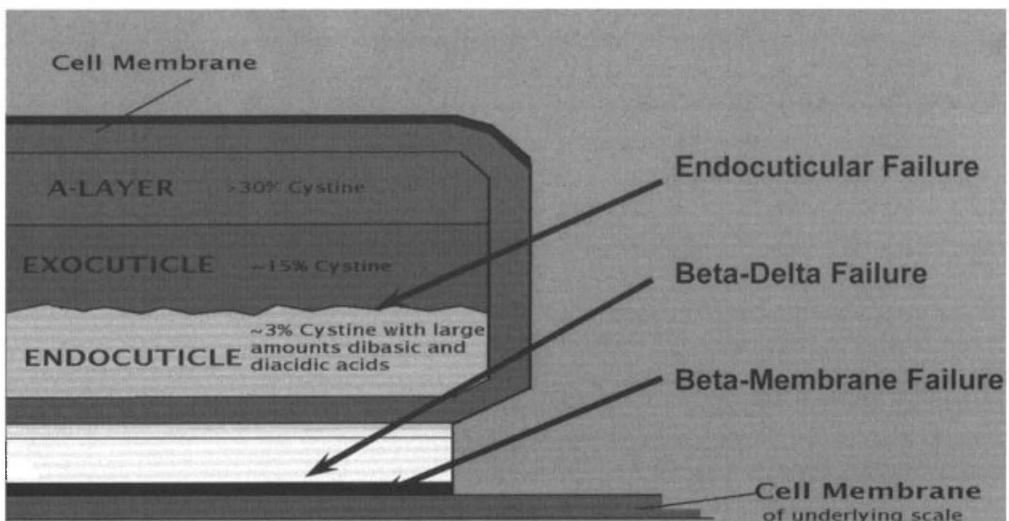


Figure 11. Schematic of the structure inside cuticle cells and the CMC using the monolayer model.

ticular layer, the major area of swelling in the cuticula (Figure 11). Upon wet combing, because of shear forces from abrasion and cyclic extension, sections of cuticle are removed, leaving large areas of endocuticular debris on the underlying scale surface. This debris confirms endocuticular involvement in this adhesion-failure process (see Figure 12).

ENDOCUTICULAR FAILURE AT HIGH RH OR IN THE WET STATE

This type of failure, leading to scale lifting, has been reported during low-strain-rate extensions by Ruetsch and Weigmann (18) and documented by SEMs (see Figure 13) (12,18). Endocuticular failure during extension has been attributed to the development of shear stress concentrations, where the highly cross-linked, rigid, exocuticle merges with the low cross-linked, highly swollen, extensible endocuticle (see Figure 11).

Feughelman and Willis (14) in their paper on adhesion failure indicate that endocuticular failure should be reexamined. We would agree that endocuticular failure is not the norm in the dry state for undamaged hair. However, since more than one worker has shown endocuticular failure at high RH and a weakness has been shown to exist at that site on heat drying (17), failure sometimes does occur in the endocuticle during deformation. More recent evidence (19) suggests that a very slow strain rate causes shear stresses within cuticle cells and leads to endocuticular failure. Furthermore, this type of

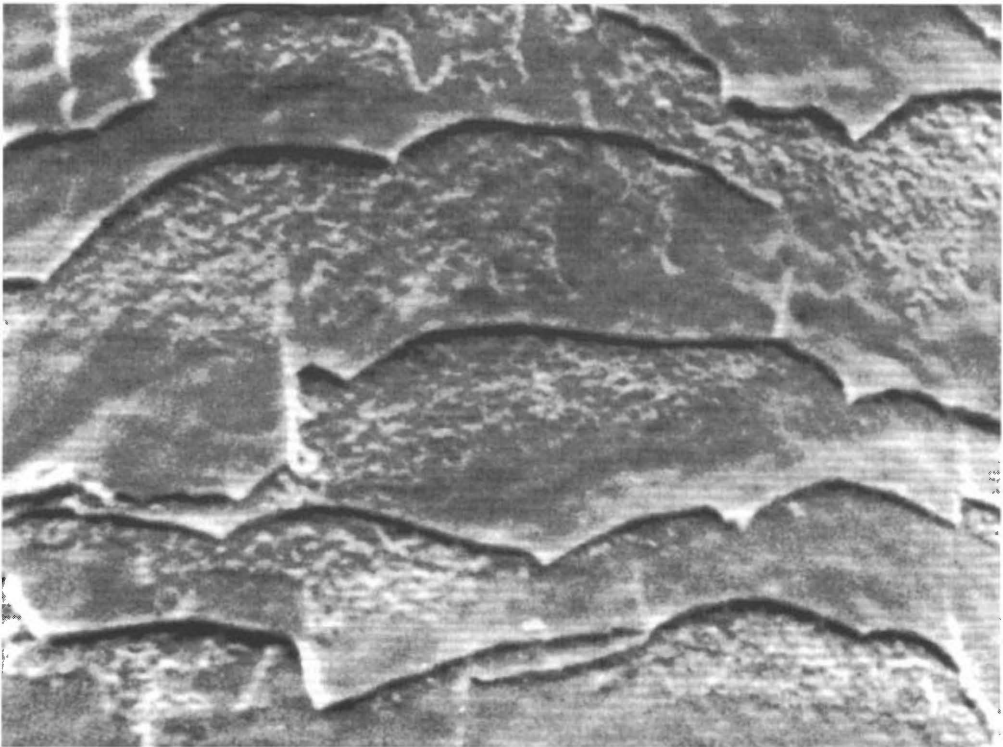


Figure 12. Hair heat-dried (as in Figure 10), then combed dry. Note exposed endocuticle structure (17). Reprinted with permission of the *Journal of the Society of Cosmetic Chemists*.

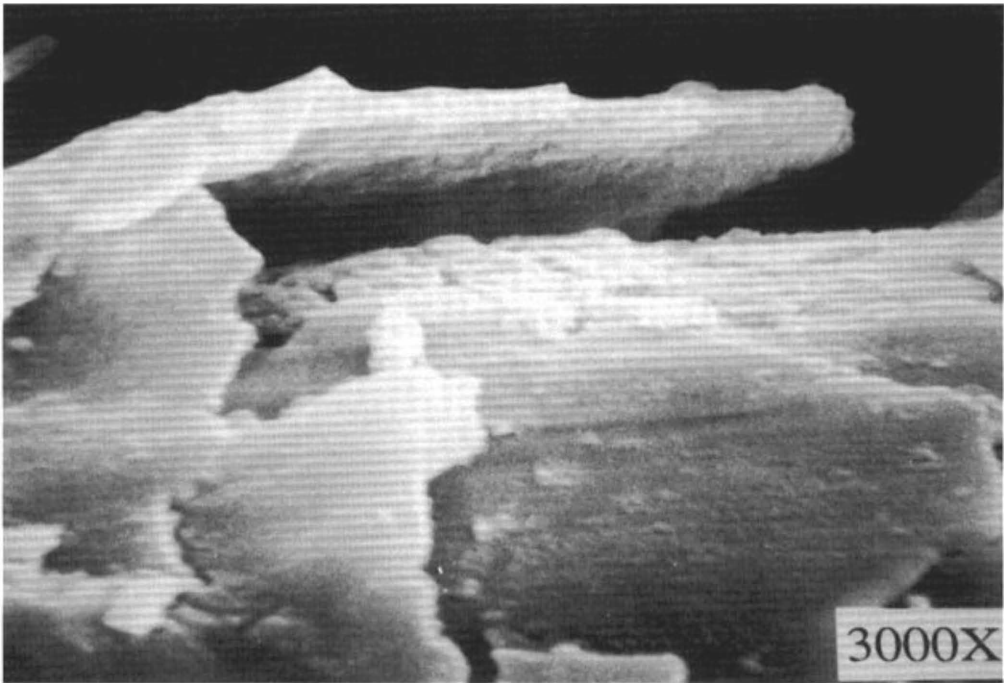


Figure 13. Endocuticular failure from stretching hair (18). Reprinted with permission of the *Journal of the Society of Cosmetic Chemists*.

failure is dependent on the RH and the extension rate and does not occur under normal tensile-loading conditions in the Instron or the Diastron (19).

CUTICLE CHIPPING IN THE WET STATE

Studies on cuticle fragmentation (20) have shown that more cuticle chipping occurs in the wet state than the dry state and that more chipping occurs as fiber swelling increases. Cuticle chipping can be induced in wet hair by combing or simply by shaking hair snippets in water or other solvents where hair fibers are rubbed against each other by the shaking action. Combing involves compression and shear stresses, while shaking fiber snippets involves shear stresses with minimal external compression forces.

In either case, a primary area of weakness is where the swollen endocuticle merges with the highly cross-linked rigid exocuticle (Figure 11) in the outermost cuticle cells that are most exposed to the extension and abrasive actions. In both cases, it is likely that endocuticular failure at a scale edge occurs first, followed by transverse cracking across a small scale segment to produce a cuticle chip. Obviously, those raised scales from prior beta-delta failure and other damaging actions in the dry state will chip off during wet rubbing. However, since combing and shaking wet hair is primarily a wet-state phenomenon, beta-delta failure is not directly involved, and in that manner wet state chipping differs from dry-state cuticle chipping. Thus, dry-state cuticle fragmentation involves failure in the cuticle-cuticle CMC, while wet-state cuticle chipping most likely involves failure inside cuticle cells.

COMBING WHILE HEAT DRYING HAIR

As indicated above, wet cuticle chipping occurs while combing and heat drying hair, but another type of fracture that we are concerned with here is depicted in Figure 14. This condition is related to the formation of deep transverse cracks across the cuticle produced by wet extension of hair or by wet extension cycling (Figure 9), but with added heat. It essentially consists of heat drying tresses and cyclic extension due to combing. The very top layers of the outermost cuticle scales shrink and become dry and brittle because moisture is driven from these layers by heat drying. Circumferential stresses from high internal swelling pressure, directly adjacent to the dry brittle layers, cause weakening in the brittle surface layers. Simultaneously, the swollen cortex exerts pressure on the cuticle-cortex CMC, initiating cracks and, at the same time, extension strains from combing act on the cuticle to propagate the cracks transversely. When these cracks approach the dry, brittle cuticle scales, the crack explodes, producing large bulbous cracks (Figure 14) instead of the uniform transverse cracks observed in extension cycling without heat (Figure 9). Ruetsch (private communication) has observed bubbles in the scale faces during thermal drying that could be precursors of these thermal cracks.

HAIR DAMAGE AND FAILURE OF ADHESION

Damage to hair generally leads to an increase in step fractures, multiple step fractures, fibrillation, and splits (16). An undamaged cuticle inhibits step fractures by interfering with fracture propagation along the fiber axis. Thus, an undamaged hair with no flaws (including no medulla) will be more likely to provide smooth fractures, especially when wet or at a high RH, than step fractures, fibrillation, or splits. Kamath and Weigmann (12) have described the formation of step fractures in two ways: After a fracture is initiated by the formation of cracks perpendicular to the fiber axis, but not completely

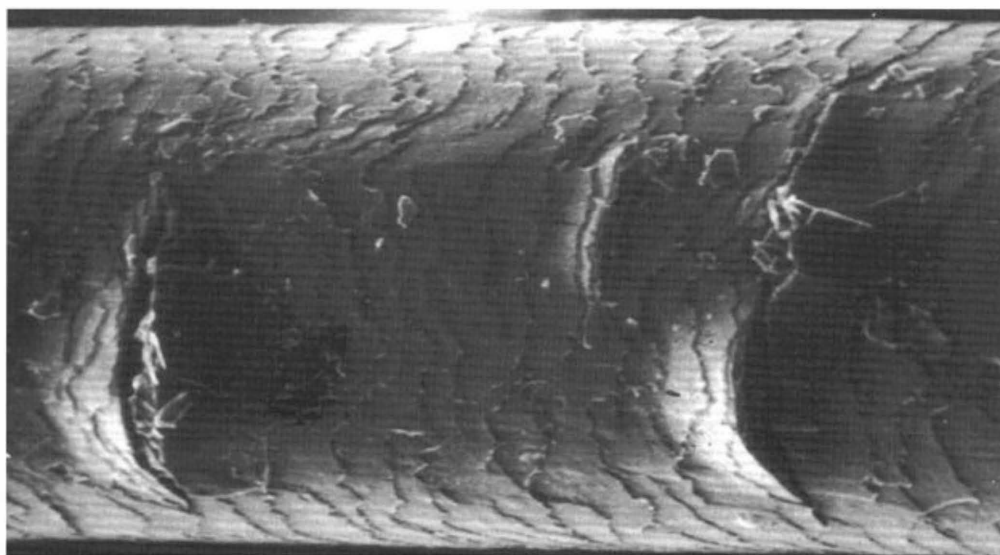


Figure 14. Deep ovoidal cracks across cuticle from simultaneously heat drying and combing wet hair (1). Reprinted with permission of Springer-Verlag.

across the fiber, stress concentrations can be relieved or diverted along the fiber axis through the intercellular cement or through flaws such as the medulla, until the stress is relieved by fiber breakage. Step fractures can also occur by simultaneous propagation of two cracks on opposite sides of the fiber, perpendicular to the fiber axis and then shearing along cortical cell surfaces through the CMC along the axis until the fiber snaps. At low RH, failure generally occurs in the CMC rather than inside cortical cells (12). At 65% RH, cortical cells may be torn apart, although this action does not appear to occur frequently.

SUNLIGHT DAMAGE

Extending hair fibers to break that have been previously damaged from disulfide bond rupture or thioester bond breakage (Figure 2) by ultraviolet light or sunlight and the consequent changes leads to multiple step fractures (see Figure 15) (Ruetsch, private communication). The entire fiber is weakened by these chemical changes, including the cortex-cortex cell membrane complex, where both axial and lateral propagation of fractures can occur more extensively than before this type of damage. This type of CMC weakening provides multiple step fractures (see Figure 15). Additional damage from extended sunlight exposure breaks peptide bonds and forms new crosslinks, leading to a very brittle fiber that provides rapidly formed smooth fractures in the periphery or the most damaged region of the fiber. Figure 15 shows smooth fracturing in the periphery (the most chemically modified part of the fiber) and multiple step fractures in the interior of the fiber.

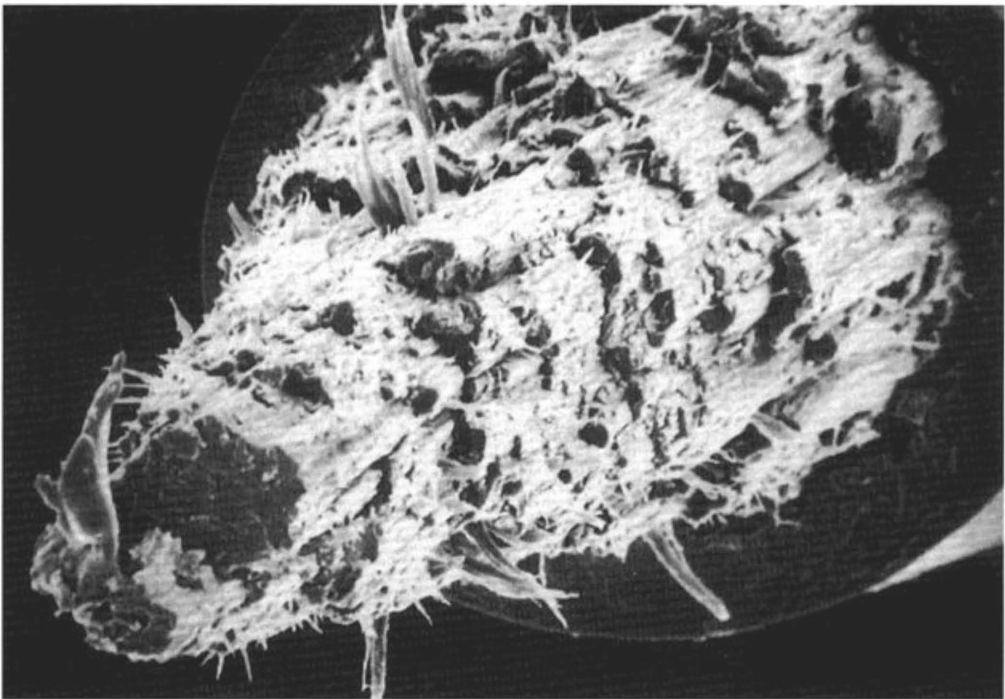


Figure 15. UV-brittle damage and “cathedral” fracture leaving smooth fracture near surface layers and multiple step fractures inside the fiber (21). Reprinted with permission of the *Journal of Cosmetic Science*.

In the final stages of sunlight damage to hair, the cuticle layers are very brittle from the chemical changes brought about by degradation of the disulfide crosslinks and the creation of crosslinks from peptide bond alterations. Because of the extensive transformations in the fiber, much cuticle differentiation disappears. Thus, in addition to failure in the fiber interior, when deformed, the fibers provide cracks in the outer brittle cuticle that are perpendicular to the fiber axis (see Figure 16).

FRACTURING OF BLEACHED AND PERMED HAIR

Hair bleaches provide damage to disulfide bonds and some damage to thioester and even to ester linkages (see the monolayer model of Figure 2B and the bilayer model of Figure 3). Bleached hair is especially sensitive to the subsequent reductive action of thioglycolate perms, leading to fibers that behave very similarly under extension to sunlight-damaged hair by providing multiple step fractures in the cortex (see Figure 17). This effect occurs because of the highly weakened cortex-cortex CMC, which provides for a much larger number of routes for the axial diversion of fractures than before these chemical changes to the fiber.

BENDING AND FAILURE OF ADHESION

Failure of adhesion caused by bending and torsional deformations needs to be examined more fully where deformation conditions, relative humidity, and hair condition are

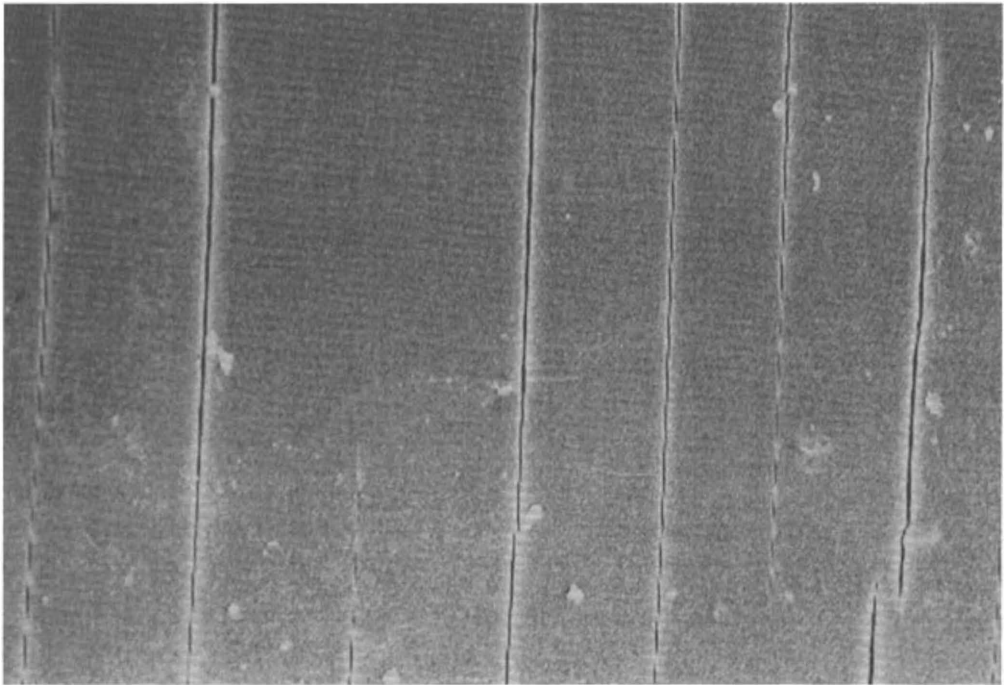


Figure 16. Sun damage leaving surface cracks perpendicular to the fiber axis upon stretching (S. Ruetsch, private communication). SEM by S. Ruetsch, TRI Princeton.

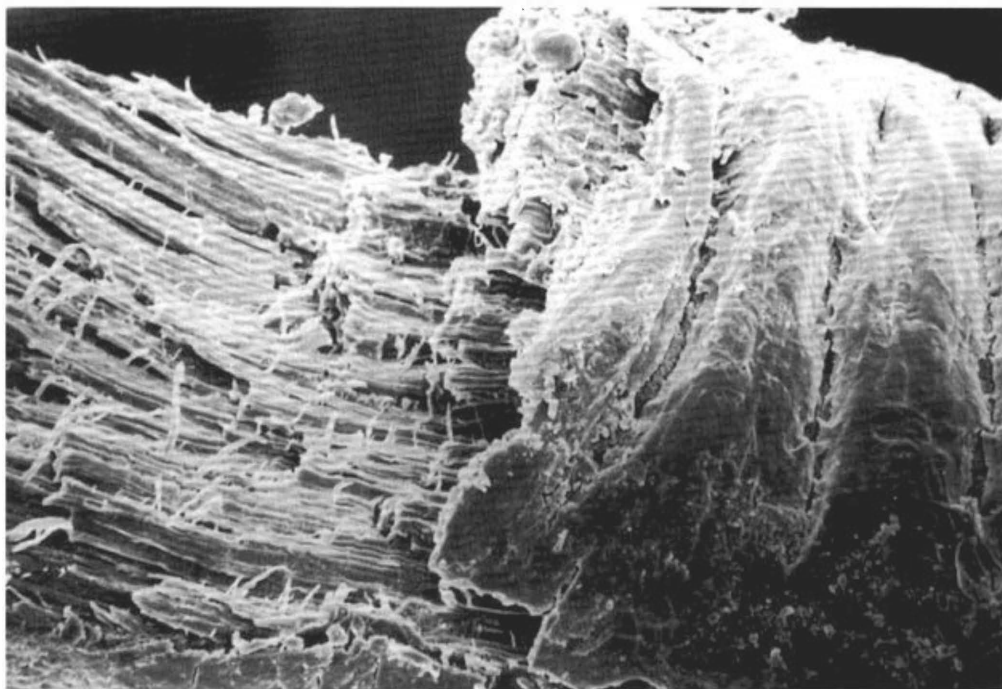


Figure 17. Multiple step fractures from stretching hair previously bleached and permed (S. Ruetsch, private communication). SEM by S. Ruetsch, TRI Princeton.

controlled systematically. Scale lifting caused by bending deformations (Figure 18) during fiber knotting, especially in the dry state, is very similar to extension-caused scale lifting and most likely involves outer beta-delta failure, particularly at low and moderate RHs. However, the condition of the hair and other variables must be considered to understand this phenomenon more fully.

ADHESION FAILURE FROM ALTERNATING TREATMENTS OF ANIONIC AND CATIONIC SURFACTANTS

Alternating treatments of anionic and cationic surfactants on hair fibers that have been previously permanent waved can provide adhesion failure and scale lifting (Figure 19). This type of failure occurs only in damaged hair, and it occurs more readily on hair that has been permanent waved on the head and stressed by shampooing, combing, and other stresses that are normally encountered in everyday wear. Hair permed in the laboratory can also provide this effect, but not to the same degree. Hair permed in the laboratory and then combed responds more than uncombed and permed hair, but still not to the same degree as hair permed on the head and exposed to normal wear and weathering actions. Extensively bleached hair can also provide this type of response, but not to the same degree as hair permed on the head. Undamaged hair does not provide this type of adhesion failure.

As indicated above, alternating treatments of anionic and cationic surfactants can produce this effect. In this reaction, anionic surfactants that are larger and more hydrophilic

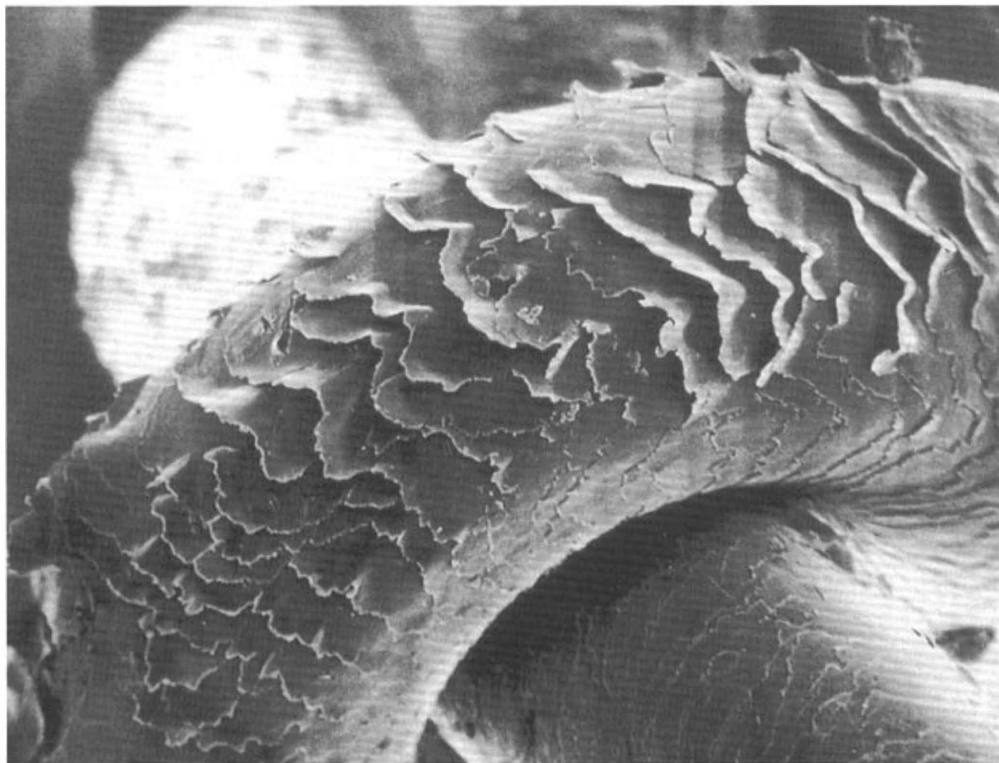


Figure 18. Bending and failure of adhesion by knotting a hair fiber (1). Photograph reproduced with permission of Springer-Verlag.

such as sodium deceth-3 sulfate or sodium laureth-3 sulfate do not produce this failure as readily as TEA dodecyl sulfate or sodium lauryl sulfate. Cationic surfactants such as cetrimonium chloride and benzalkonium chloride provide the most failure and scale lifting when applied alternately with salts of lauryl sulfate. In general, those combinations of anionic and cationic surfactants that most readily form water-insoluble cationic-anionic complexes produce the most failure, while those that do not tend to form water-insoluble complexes, such as sodium deceth-3 sulfate with tetradecyl trimonium chloride, do not tend to produce this failure.

Since permanent waving is essentially a prerequisite to this condition, and perming produces this effect more readily than bleaching, we believe that cleavage of the thioester linkage between the outer beta layer and the hydrophobic keratin protein (Figure 2B) is involved because this bond has been shown to be sensitive to strong nucleophiles like amines and mercaptans (3,22) (see Figure 20). This reaction of TGA in the cuticle-cuticle CMC weakens the outer beta layer by cleaving thioester linkages through nucleophilic displacement with TGA, creating a hydrophilic structure from a hydrophobic one at the cuticle membrane region of fatty acid attachment (hydrophobic keratin protein (b) in Figure 2A) rather than at the delta layer attachment (see the monolayer model in Figures 2B and 20). The next step involves penetration of cationic surfactant into this site and bonding by salt linkage to the newly formed anionic sites in this weakened region. Subsequently, anionic surfactant penetrates and reacts to form an

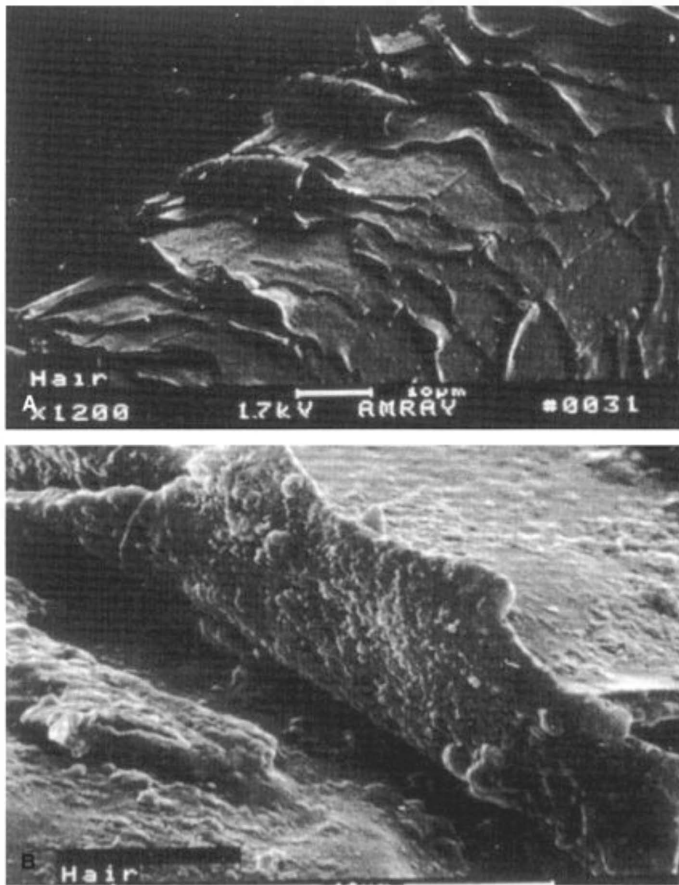


Figure 19. Adhesion failure from alternating treatments with anionic and cationic surfactants on previously permed hair (1). Photographs reproduced with permission of Springer-Verlag.

insoluble complex with the sorbed cationic surfactant (Figure 21). After several repetitive anionic-cationic reactions, buildup of a hydrophobic anionic-cationic complex occurs, producing adhesion failure at this site. These reactions will produce beta-membrane failure rather than beta-delta failure (see Figure 11).

The fact that scale lifting occurs from the reaction of TGA-treated hair more readily than with bleached hair suggests that this effect is governed more by reaction with the thioester linkage than with the disulfide grouping. Nevertheless, the net effect is to weaken the CMC and increase its tendency to fail under stress.

SUMMARY AND CONCLUSIONS

Although adhesion failure in hair fibers can occur inside cuticle and cortical cells, it frequently occurs in the cell membrane complex (CMC), often involving interlayer bonding. Therefore, a model of the CMC is presented, based on prior research in which we propose interconnecting bonds between the different layers to assist in our interpretation of hair fracturing mechanisms for cuticle chipping, deep transverse cuticle cracks,

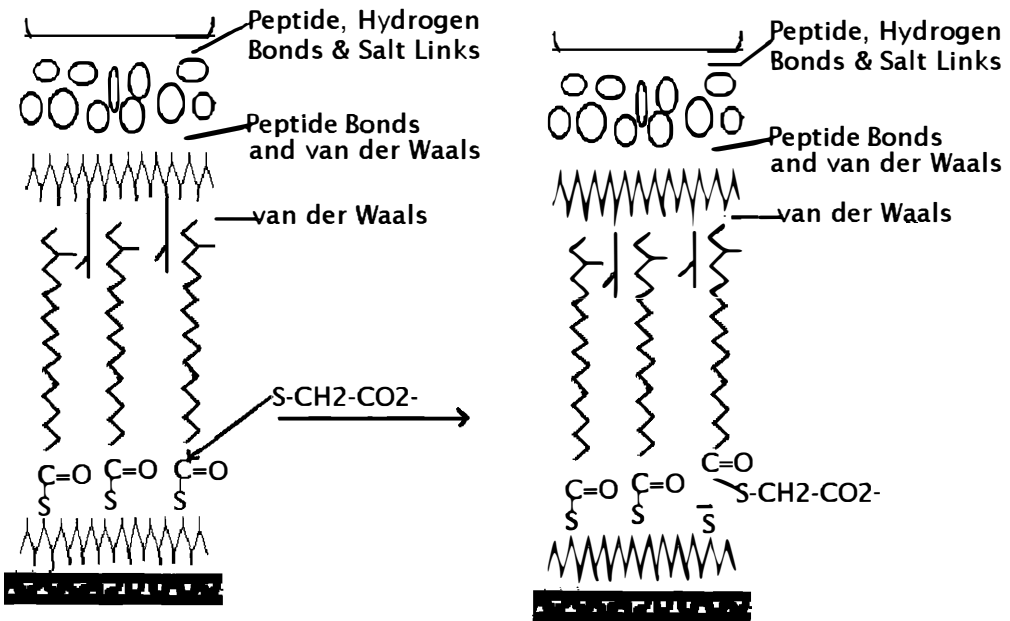


Figure 20. Reactions of the thioester linkage with TGA in the upper beta layer.

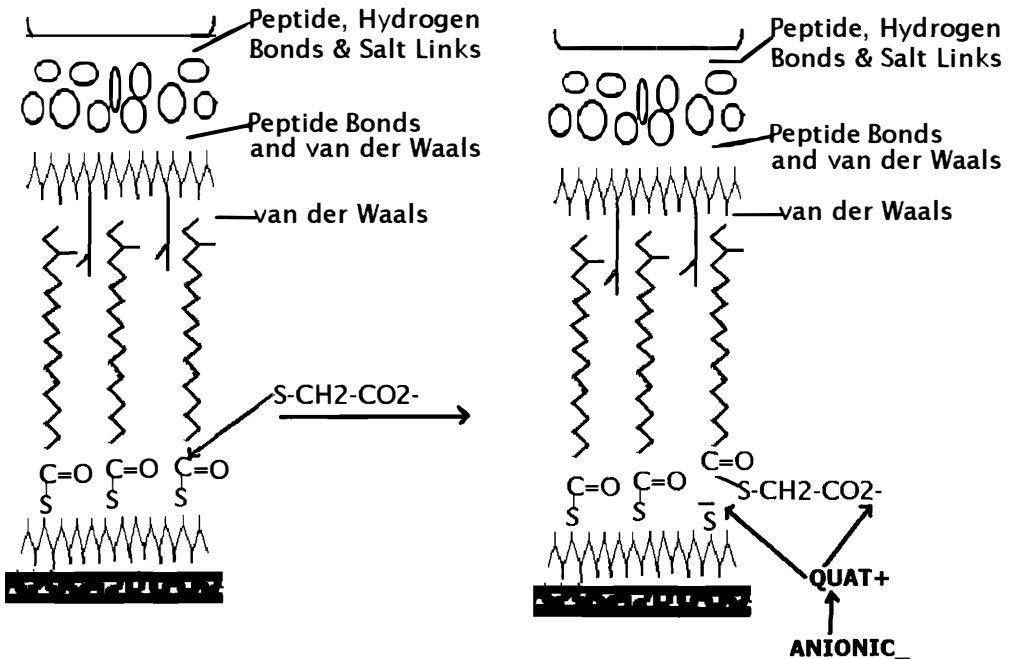


Figure 21. Reactions of thioester with TGA followed by quat & anionic in this same layer.

cracks during heat drying, scale lifting by surfactants, and catastrophic failure. Failure in the wet state generally involves hydrophilic layers (endocuticle or the contact zone of the CMC) or bonding between hydrophilic layers, whereas failure in the dry state

generally involves bonding between hydrophobic layers (beta-delta failure). Chemical damage by permanent waves, bleaches, and sunlight can influence the initial sites of failure and increase the number of possible routes for crack propagation, thus leading to more complex fracture patterns.

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