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Abnormal isoaspartyl residues in erythrocyte membranes from psoriatic patients

- Stefania D'Angelo,
- Serena Lembo,
- Filomena Flora,
- Maria Luigia De Bonis,
- Anna Balato,
- Fabio Ayala,
- Nicola Balato,
- Patrizia Galletti,
- Vincenzo Zappia

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Abstract

Spontaneous protein deamidation of labile asparagines (Asn), generating abnormal isoaspartyl residues (IsoAsp), is associated with cell aging and enhanced by an oxidative microenvironment. The presence of isopeptide bonds impairs protein structure/function and can trigger autoimmune responses. To minimize the damage, IsoAsp can be "repaired" by a specific l-isoaspartate-(d-aspartate)-protein-*O*-methyltransferase. The condition of chronic oxidative stress reported in psoriatic patients, and the potential etiological role of unknown self-antigens, prompted us to investigate Asn deamidation in psoriatic tissues. Erythrocytes (RBC) were selected as the model system since, lacking protein synthesis apparatus, they are unable to replace damaged proteins. Blood samples were obtained from 36 patients and 34 controls. l-isoAsp content was highly increased in RBC membrane proteins from psoriatic patients. Deamidated species included ankyrin, band 4.1, band 4.2 and the integral membrane protein band 3. A functional analysis demonstrated that this result was unrelated to a reduced efficiency of the *S*-adenosylmethionine-dependent repair system suggesting an increased protein instability at Asn sites, responsible for IsoAsp accumulation in psoriatic patients.