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# **Product Information**

## Anti-Lamin A-Atto 647N

produced in rabbit, affinity isolated antibody

Product Number L3544

# **Product Description**

Anti-Lamin A is produced in rabbit using as immunogen a synthetic peptide corresponding to a fragment of human lamin A (Gene ID: 4000) conjugated to KLH. The product is prepared by conjugation of the affinity purified Anti-Lamin A antibody to Atto 647N NHS ester (Abs<sub>max</sub> = 644 nm;  $Em_{max}$  = 673 nm) (Product Number 18373), and the conjugate is purified by gel filtration to remove unbound Atto 647N-NHS fluorophore.

Anti-Lamin A-Atto 647N recognizes human Lamin A by direct immunofluorescence.

Lamin A is a structural protein of the nuclear lamina. The nuclear lamina is a meshwork of intermediate filaments that underlies the inner face of the nuclear envelope. The major components of the nuclear lamina are the lamins that may be classified into two types, A and B. Both A- and B-type lamins are characterized by an  $\alpha$ -helical rod domain to enable assembly into filaments, a nuclear localization sequence, and a carboxy-terminal CAAX box isoprenylation sequence for nuclear membrane targeting.  $^2$ 

A-type lamins, lamin A and lamin C, are products of a single gene, *LMNA*, which are produced by alternative splicing, resulting in proteins of 664 and 572 amino acids, respectively.<sup>3</sup> The first 566 amino acids of Lamin A and C are identical. Prelamin A, the precursor of Lamin A, has 98 unique amino acids and is farnesylated at its carboxy terminus after synthesis. The last 18 amino acids, which contain the farnesyl group, are removed by an endoproteolytic cleavage, producing the mature Lamin A.<sup>4</sup>

Lamins are expressed in most somatic cells. They interact with integral proteins of the inner membrane of the nuclear envelope, such as LAPs 1 and 2 (lamina-associated polypeptides), LBR (Lamin B receptor), and emerin. They also interact with chromatin and nuclear pore complexes. 4

Lamin A is cleaved into a 47 kDa fragment during apoptosis. Lamin A cleavage seems to be essential for chromatin condensation and nuclear disassembly in apoptosis. A Mutations in Lamin A and C have been linked to a variety of rare human diseases including muscular dystrophy, lipodystrophy, cardiomyopathy, neuropathy, and progeroid syndromes (collectively termed laminopathies) and to premature aging (Hutchinson-Gilford progeria syndrome). Most diseases arise from dominant, missense mutations.

### Reagent

The product is provided as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as preservative.

Antibody concentration: 1.5–3.0 mg/mL

Molar Ratio (F/P): 2-9

# **Precautions and Disclaimer**

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

### Storage/Stability

For continuous use, store at 2–8 °C for up to one month. For extended storage, freeze in working aliquots. Protect from prolonged exposure to light. Repeated freezing and thawing is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

# **Product Profile**

<u>Direct Immunofluorescence</u>: a working concentration of 5–10 μg/mL is recommended using human HeLa cells.

Note: Appropriate filter ( $Abs_{max} = 644 \text{ nm}$ ;  $Em_{max} = 673 \text{ nm}$ ) should be used in order to observe the fluorescence signal.

<u>Note</u>: In order to obtain best results in various techniques and preparations, it is recommended to determine optimal working dilutions by titration.

# References

- 1. Aebi, U. et al., *Nature*, **323**, 560-564 (1986).
- 2. Rao, L. et al., *J. Cell Biol.*, **135**, 1441-1455 (1996).
- 3. Lloyd, D.J. et al., *Hum. Mol. Gen.*, **11**, 769-777 (2002).
- 4. Worman, H.J., and Courvalin, J.C., *Trends Cell Biol.*, **12**, 591-598 (2002).
- 5. Schirmer, E.C. et al., *J. Cell Biol.*, **153**, 479-489 (2001).
- 6. Ruchaud, S. et al., *EMBO J.*, **21**, 1967-1977 (2002).
- 7. Worman, H.J., and Courvalin, J.C., *J. Clin. Invest.*, **113**, 349-351 (2004).
- 8. Plasilova, M., et al., *J. Med. Genet.*, **41**, 609-614 (2004).

VS,ST,KSS,KAA,PHC,MAM 01/19-1