

Background

Heart-type fatty acid binding protein (hFABP) also known as mammary-derived growth inhibitor is a protein that in humans is encoded by the *FABP3* gene.^[1] *FABP3* gene contains four exons and its function is to arrest growth of mammary epithelial cells. This gene is also a candidate tumor suppressor gene for human breast cancer. H-FABP is a small cytoplasmic protein (15 kDa) released from cardiac myocytes following an ischemic episode.^[2] Like the nine other distinct FABPs that have been identified, H-FABP is involved in active fatty acid metabolism where it transports fatty acids from the cell membrane to mitochondria for oxidation.^[2]

H-FABP is a sensitive biomarker for myocardial infarction^[3] and can be detected in the blood within one to three hours of the pain. H-FABP is 20 times more specific to cardiac muscle than myoglobin,^[4] H-FABP is recommended to be measured with troponin to identify myocardial infarction and acute coronary syndrome in patients presenting with chest pain. H-FABP measured with troponin shows increased sensitivity of 20.6% over troponin at 3-6 hours following chest pain onset.^[5] Its rapid release into plasma after myocardial injury - 60 minutes after an ischemic episode,^[6] and its relative tissue specificity. Measuring H-FABP in combination with troponin increased the diagnostic accuracy and with a negative predictive value of 98% could be used to identify those not suffering from MI at the early time point of 3-6 hours post chest pain onset.^[5] The effectiveness of using the combination of H-FABP with troponin to diagnose MI within 6 hours is well reported.^[6] H-FABP also has prognostic value. Alongside D-dimer, NT-proBNP and peak troponin T, it was the only cardiac biomarker that proved to be a statistically significant predictor of death or MI at one year. Patients who were TnI negative but H-FABP positive had 17% increased risk of all cause mortality within one year compared to those patients who were TnI positive but H-FABP negative.^[7] H-FABP has been proven to significantly predict 30 day mortality in acute pulmonary embolism.^[8] H-FABP is more effective than Troponin T in risk stratifying Chronic Heart Failure patients.^[9]

References

1. Phelan CM, et al. (1996). *Genomics* **34** (1): 63–8.
2. Kleine AH, et al. (1992). *Molecular and Cellular Biochemistry* **116** (1-2): 155–62.
3. Tanaka T, et al. (1991). *Clinical Biochemistry* **24** (2):
4. Ghani F, et al. (2000). *Clinical Chemistry* **46** (5): 718–9.
5. Glatz JF, et al. (1994). *British Heart Journal* **71** (2): 135–40.
6. Van Nieuwenhoven FA, et al. (1995). *Circulation* **92** (10): 2848–54.
7. Viswanathan K, et al. (2010). *Journal of the American College of Cardiology* **55**: 2590–8.
8. Kaczyńska A, et al. (2006). *Clinica Chimica Acta; International Journal of Clinical Chemistry* **371** (1-2): 117–23. .
9. Niizeki T, et al. (2007). *Journal of Cardiac Failure* **13** (2): 120–7.



Genorise® Recombinant Human H-FABP

Catalog Number: GR1119002

Description

Size: 10 µg

Source: *E coli* derived

Component: Met1 –Ala133

Accession # P05413

Predicted Molecular Mass: 15 kDa (monomer)

Specifications

SDS-PAGE: 15 kDa, reducing conditions

Purity: >97%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation: Lyophilized from a 0.2 µm filtered PBS with BSA as a carrier protein.

Preparation and Storage

Reconstitution: Reconstitute at 100 µg/mL in sterile PBS.

Shipping: The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage: Use a manual defrost freezer and avoid repeated freeze thaw cycles.

- 6 months from date of receipt, -20 to -70°C as supplied.
- 3 months, -20 to -70°C under sterile conditions after reconstitution.

DECLARATION

THIS REAGENT IS FOR IN VITRO LABORATORY TESTING AND RESEARCH USE ONLY. DO NOT USE IT FOR CLINICAL DIAGNOSTICS. DO NOT USE OR INJECT IT IN HUMANS AND ANIMALS.

**FOR LABORATORY RESEARCH USE ONLY
NOT FOR USE IN HUMANS AND ANIMALS**