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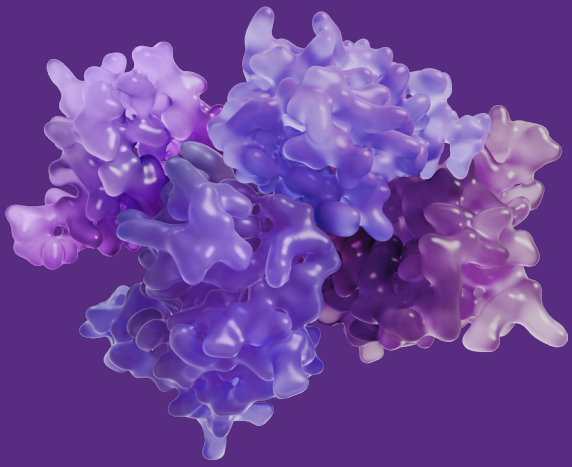
Prescribing information and Adverse Event Reporting can be found at the bottom of the page

BEYONTTRA® ▼
(acoramidis) 356mg tablet

Transthyretin (TTR) explained



Beyonttra® ▼ is indicated for the treatment of wild-type and variant transthyretin amyloidosis in adults with cardiomyopathy (ATTR-CM).[1]

Adverse event reporting and Prescribing information for Beyonttra® ▼ (acoramidis) is available by scanning or clicking the QR code on the left

Scan or click the QR code for prescribing information and adverse event reporting information.

For direct access to this prescribing information,please ensure that your device's browser

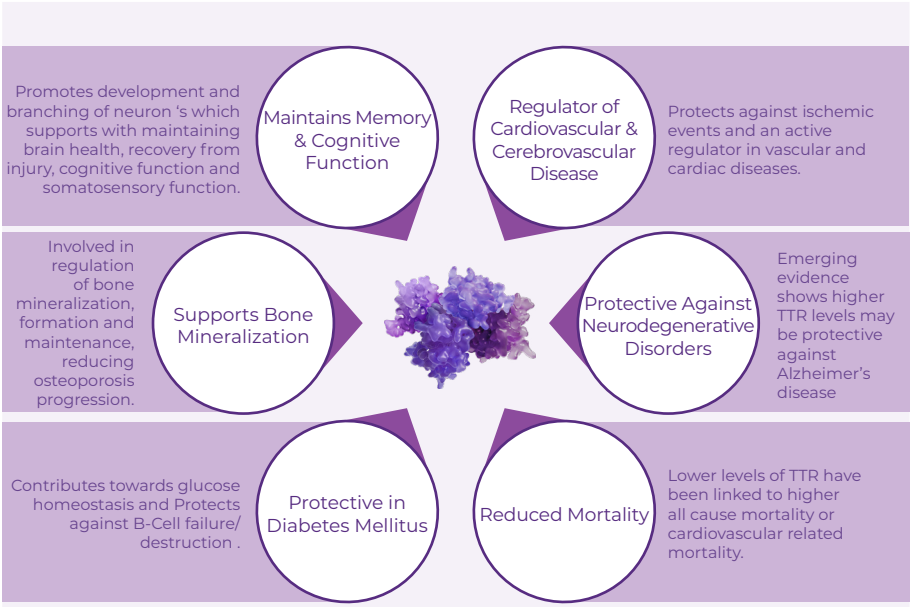
Reporting adverse events and quality complaints
Adverse events should be reported. Reporting forms and information can be found at https://yellowcard.mhra.gov.uk or search MHRA Yellow Card in Google Play or Apple App Store. Adverse events should also be reported to Bayer plc.
If you want to report an adverse event or quality complaint, reports can be directed to Tel: 0118 206 3500 or Email: pvuk@bayer.com
Further information is available on the "contact" tab at www.bayer.co.uk.

PP-BEY-GB-0258
Date of preparation: May 2026

What is transthyretin (TTR)?

TTR is a **highly conserved transport protein** made up of four monomers, primarily produced in the liver, and in other organs such as the brain (choroid plexus) and eyes (retinal pigment epithelium)[2]

TTR: A vital protein that has diverse and emerging roles throughout the body, it is responsible for supporting the healthy functionality of multiple organ systems



Adapted from Liz MA, et al 2020; and Gertz MA, et al 2025.[2,3]

TTR is also a crucial protein in ageing populations[2]

TTR-mediated vitamin A transport is **essential for visual function**, including **night vision**.[4]

Emerging evidence supports the neuroprotective role in Alzheimer's disease and its **potential systemic relevance in age-associated pathologies**[5]

Transthyretin amyloid cardiomyopathy (ATTR-CM)

ATTR-CM is a progressive and fatal cardiac disease caused by the destabilisation of the TTR tetramer[6–8]

- TTR destabilisation can be caused by a genetic mutation in the TTR gene (variant ATTR-CM) or related to the ageing process (wild-type ATTR-CM)[9,10]
- Dissociated monomers can misfold and aggregate, leading to the **formation of amyloid fibrils** that build up in tissues throughout the body, including the heart[2,11,12]
- Amyloid fibril accumulation can lead to diverse cardiac and extracardiac symptoms including **heart failure**[2,11,12]

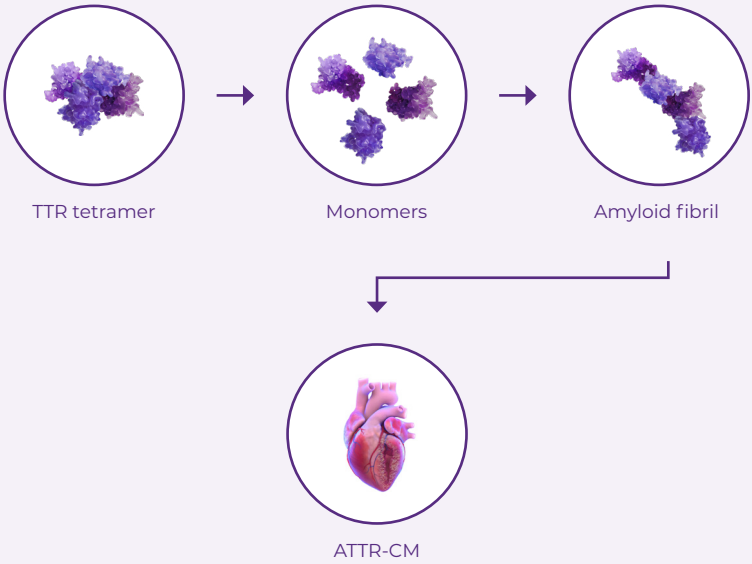


Image design by Bayer

Beyonttra is the only stabiliser with near-complete (≥90%) TTR stabilisation in the label[1]

TTR ↑ Serum TTR was higher in the Beyonttra group compared with placebo, from day 28 through to month 30.*[1]

EARLY BENEFITS

Patients on Beyonttra can experience improvements in all-cause mortality first cardiovascular-related hospitalisation (CVH) as early as **3 months** versus placebo[13,14]

HEART PROTECTION

Reduced the risk of death or first CVH versus placebo by

35.5%[1, 13,14]
Relative risk reduction (RRR)
(HR: 0.645,95% confidence interval [CI]: 0.500-0.832, p=0.0008)

14.6%
Absolute risk reduction (ARR)

MORE FREEDOM

Reduced annual frequency of CVH versus placebo

50.4%[14,15]
RRR
(Relative rate ratio: 0.496,95% CI: 0.355-0.695, p<0.0001)

26%
ARR

* Mean 9.1 mg/dL in serum TTR levels within 28 days which was sustained throughout the 30-month treatment period.[1]

ACM, all-cause mortality; **ATTR-CM,** transthyretin amyloid cardiomyopathy; **CI,** confidence interval; **mITT,**modified intention-to-treat; **NT-proBNP,** N-terminal pro-B-type natriuretic peptide; **TTR,** transthyretin.

References: 1. Beyonttra UK Summary of Product Characteristics. **2.** Liz MA, et al. Neurol Ther. 2020;9(2):395–402; **3.** Gertz MA, et al. Annals of Medicine. 2025;57(1)2536755; **4.** Steinhoff JS, et al. Nutrients. 2022;14(6):1236; **5.** Corino C, et al. Mol Neurobiol. 2025;62(3):2945–2954; **6.** Jain A, Zahra F. Transthyretin Amyloid Cardiomyopathy (ATTR-CM). In: StatPearls. StatPearls Publishing,Treasure Island (FL); 2025. PMID: 34662045; **7.** Gonzalez-Lopez E, et al. JACC CardioOncol. 2022;4(4):442–454; **8.** Gonzalez-Duarte A, Ulloa-Aguirre A. Int J Mol Sci . 2021;22(23):13158; **9.** Ruberg FL, et al. J Am Coll Cardiol. 2019;73(22):2872–2891; **10.** Kittleson MM, et al. Circulation. 2020;142(1):e7–e22; **11.** Vieira M, Saraiva MJ. Biomol Concepts. 2014;5(1):45–54; **12.** Witteles RM, et al. JACC Heart Fail. 2019;7(8):709–716; **13.** Judge DP, et al. Circulation. 2025;151(9):601–611 (inc. supplement); **14.** Judge DP, et al.JACC. 2025;85(10):1003–1014; **15.** Gillmore JD, et al. N Engl J Med. 2024;390:132–142

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