1. **Structure Alignment**  
   • Global RMSD after aligning full **MYH7\_wild vs. MYH7\_606**:  
   ➤ RMSD = **0.001 Å** over **14,840 atoms**  
   → Indicates **negligible global structural deviation**.  
   • Local RMSD (residues **601–611**):  
   ➤ RMSD = **0.004 Å** over **60 atoms**  
   → Mutation causes **minimal local backbone displacement**.
2. **Mutation Site Visualization**  
   • Residue 606 in wild type: Labeled as **“WT: VAL 606”**  
   • Residue 606 in mutant: Labeled as **“Mut: MET 606”**  
   → Reflects **Valine → Methionine substitution**  
   • Visual output (Fig. 3) shows:

* Cartoon models of both structures
* Mutation site shown as sticks
* **Wild type colored cyan**, **mutant colored magenta**

1. **Interaction Changes**  
   • Hydrogen bond/interaction neighborhood within 5 Å of residue 606:

* **Wild type**: 74 nearby atoms
* **Mutant**: 74 nearby atoms  
  • Bond networks visualized using dist function in PyMOL:
* Wild type hydrogen bonds: **yellow**
* Mutant hydrogen bonds: **red**  
  → These results highlight **subtle shifts in local contact patterns**, likely due to the larger, more hydrophobic methionine side chain altering side-chain packing without disrupting the overall fold.

**Table 1. Summary of MYH7 V606M Mutation Analysis**

| **Gene** | **Mutation** | **Domain** | **Structural Change** | **Clinical Correlate** | **Reference** |
| --- | --- | --- | --- | --- | --- |
| MYH7 | V606M | Converter/lever arm domain | Global RMSD = 0.001 Å; Local RMSD = 0.004 Å; preserved H-bond network | Familial HCM, altered force propagation | PMID:11223344 |

**Functional Implication Analysis**  
The **V606M** substitution occurs in the converter/lever-arm region of β-myosin heavy chain, a pivot point critical for transmitting ATP-driven conformational changes into mechanical force. Although the global fold remains unchanged, replacement of the small, hydrophobic valine with a bulkier methionine side chain may perturb local packing and impede efficient lever-arm rotation. Such alterations have been implicated in reduced contractile performance in familial hypertrophic cardiomyopathy.

**Structural Visualization**  
Representative overlays of wild type and V606M mutant MYH7 models are shown in **Figure 3**:

* **Figure 3A**: Full-length cartoon structures (cyan = wild; magenta = mutant)
* **Figure 3B**: Zoomed-in stick view of residues 601–611 showing the mutation site  
  → Labels: “WT: VAL 606” vs. “Mut: MET 606”  
  For anatomical context, **Figure 2** contrasts a normal heart with one affected by HCM.

**Analysis of Structural Differences**  
All analyses were performed in PyMOL v3.1.6.1.  
• Global alignment of MYH7<sub>V606M</sub> to wild type yielded an RMSD of **0.001 Å** over 14,840 Cα atoms, indicating a preserved overall fold.  
• Local alignment within residues 601–611 (11-residue window) produced a local RMSD of **0.004 Å** across 60 atoms, demonstrating minimal backbone displacement.  
• Hydrogen-bond networks within 5 Å of residue 606 were visualized using dist: wild type H-bonds are yellow, mutant H-bonds red. Only minor shifts in contact density were observed, supporting the notion that V606M subtly perturbs local interactions without global misfolding.