

Table 1 Primers used for amplification of fragments from *TNNT2*

Exon	Forward (5'–3')	Reverse (5'–3')
2	GAGCTTCTCTGAGGAAGGCA	CTACCCAGAATCCGAGGGAC
3 and 4	CAGGGCAGCGTGGACTCCC	CCCAGGGCTCCCAGGATTT
5	CCATTCTCTGCTCTGGGTTC	GTGCACATGGGAAAGCTGTTCT
6	TAGGGCTTATCTGTGGGAAGGC	CTTCCCTGAAAAGAGCACTG
7	GAAATGAAAATCCACAGGA	TACTGCACCCCGTTCATCA
8 and 9	CTCTAGGAAGGATCAGGGCCC	CTCACAAAAGGGATGGAGGA
10	GCGATGTCACCTTCTCCCTA	CACCGCACCCGGCCAATA
11	GGTTTCCAATCCTTTCCCTAA	GCTGCAGTGGACACCTCATT
12	GCCTTTGTCTTCTGCCTTCTC	CAGCCAGCCCAATCTCTTCACT
13	ACAGGGAGGGGGCAATCTGGCC	CCCAGAGCAGATGCGGGCAGTG
14	ACTGGGTGCTGCCGTCTGGTC	AAGGGGGCTGTTGGGGAATAGG
15	CACTCAGCCCCCTTCTCC	AAGCTTCTCCGCCACATTT
16	GGCACCCAGTCTACCC	GTCCCCCTCAACAGCACTTT

The PCR conditions of these genes were modified and fragments were analyzed by electrophoresis, as described previously [36, 37].

Proband patients bearing *TNNT2* mutations were additionally examined for reported HCM mutations, the coding regions of *MYH7*, *MYBPC3*, *TNNI3*, α -tropomyosin, *MLY2*, *MLY3*, and all exons, and a promoter region of the α -galactosidase A gene. Primer sequences and detailed PCR conditions for these additional analyses are available upon request. Reference sequences and single-nucleotide polymorphism information were obtained from the National Center for Biotechnology Information.

Results

Clinical report and DNA analysis

To investigate mutations of *TNNT2*, we performed DNA analysis in 173 unrelated Japanese patients with familial HCM. We identified three reported mutations and a new mutation of *TNNT2* in 11 individuals from four families (A, B, C, D) (Table 2).

In family A, all five members who carried or were suspected of having HCM also had arrhythmia. Four of them had blocks, including a complete atrioventricular block (CAVB) and complete right bundle branch block (CRBBB) (Fig. 1; II-1, 6, III-1, 2), and one had bradycardia. Two of them died suddenly (Fig. 1; I-2, III-1), and one was a ventricular fibrillation (Vf) survivor (Fig. 1; III-2). The proband (Fig. 1; III-2), aged 12 years, with sinus rhythm (SR) and a CRBBB, had an episode of Vf, and her elder brother (Fig. 1; III-1), aged 14, also with SR and a CRBBB, had died suddenly. Her father and her uncle had pacemakers implanted to treat complete AV blocks. Her

mother and mother's family had no symptoms or abnormal findings. The proband and her brother and father (Fig. 1; III-2, III-1, I-2) showed mild LVH (maximum wall thickness <20 mm). All three members underwent cardiac catheterization, and showed significant left ventricular relaxation abnormalities.

DNA analysis showed that the proband and her elder brother had the double reported mutations, Arg130Cys and Phe110Ile, of *TNNT2* (Fig. 1). Her father, who had arrhythmia and mild HCM, had the Phe110Ile mutation, and her mother (II-6'), who had no symptoms or abnormal findings, had the Arg130Cys mutation.

In family B, 3 of 10 members who carried or were suspected of having HCM were thought to have obstructive hypertrophic cardiomyopathy. Two female members (Fig. 2; II-4, III-9) died suddenly at a young age. The proband (Fig. 2; III-7) showed apical hypertrophy and apical aneurysm at a comparatively young age. Her aunt (Fig. 2; II-4), who had a heart murmur, was thought to have hypertrophic obstructive cardiomyopathy (HOCM), and died when she was 19 years old. Her younger sister (Fig. 2; III-9) did not show obstructive HCM, but died suddenly when she was 21 years old. Her grandmother (Fig. 2; I-1) died suddenly at the age of 50 years. DNA analysis showed that the proband (Fig. 2; III-7), her son (Fig. 2; IV-3), and her cousin (Fig. 2; III-2), who carried HCM, had an Arg92Trp mutation of *TNNT2*.

In family C, the proband (Fig. 3; IV-3), her mother (Fig. 3; III-3), and her grandmother (Fig. 3; II-3) showed mild cardiac hypertrophy (maximum wall thickness <20 mm) and asymmetric septal hypertrophy, and her myocardial hypertrophy had been gradually increasing. Her mother was suspected of having a shifting dilated phase by recent cardiac magnetic resonance imaging (MRI). The proband (Fig. 3; IV-3) had symptomatic West syndrome following perinatal

Table 2 Clinical features of patients with *TNNT2* mutation in families A, B, C, and D

HCM individuals	A III-2	A III-1	A II-6	A II-6' (III-2/ mother)	B III-7	B III-2	B IV-3	C IV-3	C III-3	C II-3	D III-2
Exon	9 and 10	9 and 10	9	10	9	9	9	10	10	10	8
Nucleotide substitution	T328A and C388T	T328A and C388T	T328A	C388T	C274	C274	C274	C388	C388	C388	T236C
Amino substitution	P110I and R130C	P110I and R130C	P110I	R130C	R92W	R92W	R92W	R92W	R92W	R92W	I79T
Sex	F	M	M	F	F	M	M	F	F	F	F
Age (years)	12	14	55	46	35	41	2	42	15	80	25
Outcome (years)	12, Vf survivor	14, died suddenly									24, Vf survivor
Clinical diagnosis	HCM	HCM	CAVB, PMI	Normal	DHCM (HCM)	Abnormal ECG	HCM	HCM	HCM	HCM	HCM
Electrocardiogram	SR + CRBBB	SR + CRBBB, abnQ	PM rhythm (CAVB)	WNL	R-wave progression	Details unknown	RVH, deep Q	LVH, ST dep	Details unknown	Details unknown	ST dep, inv T
2D echocardiogram	LVH, ASH	LVH, ASH	LVH	WNL	LVH, apical hypertrophy	Details unknown	LVH	LVH, ASH	LVH	Details unknown	LVH, ASH
Cardiac catheterization	LV relaxation abnormalities	LV relaxation abnormalities	LV relaxation abnormalities	ND	ND	ND	ND	ND	ND	ND	ND

HCM hypertrophic cardiomyopathy, *HO*CM hypertrophic obstructive cardiomyopathy, *DHCM* dilated phase of HCM, *LVH* left ventricular hypertrophy, *Vf* ventricular fibrillation, *CAVB* complete atrioventricular block, *SR* sinus rhythm, *CRBBB* complete right bundle branch block, *abnQ* abnormal Q wave, *LVH* left ventricular hypertrophy, *RVH* right ventricular hypertrophy, *ST dep* ST depression, *inv T* inversion T, *PMI* pacemaker implantation, *ECG* electrocardiogram, *ASH* asymmetric septal hypertrophy, *APH* apical hypertrophy, *ND* not determined, *WNL* within normal limits

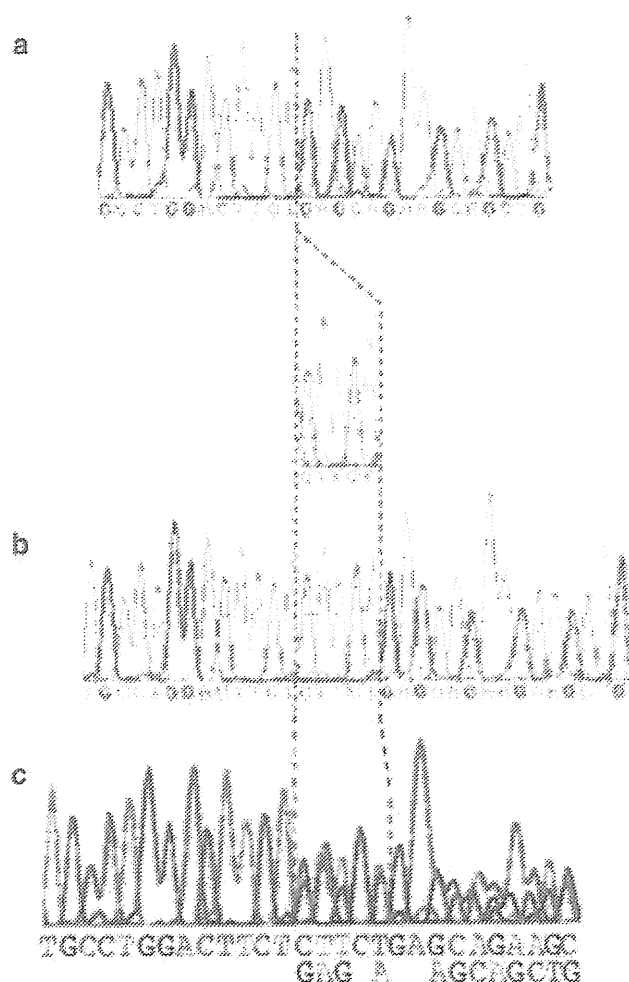


Fig. 6 a The deletion/deletion (D/D) polymorphism. b The insertion/insertion (I/I) polymorphism. c The insertion/deletion (I/D) polymorphism

(in 16 individuals of 6 Japanese Families) [19, 20]. Both of these reported pedigrees are Asian.

In family A, whereas the proband and her elder brother, who carried these two mutations, showed similar reported phenotypes of the Arg130 Cys mutation [21], her mother and mother's family members, who had the Arg130Cys mutation, showed no abnormal clinical features such as cardiac hypertrophy, sudden death, or abnormal electrocardiograms. Furthermore, her father and father's family members, who had the Phe110Ile mutation, showed atrioventricular blocks, and these phenotypes have not been reported.

In family B, an Arg92Trp mutation of *TNNT2* was found. The mutation showed comparatively slight cardiac hypertrophy or a high incidence of sudden death in males (19 individuals of two mixed racial families) [22–24]. However, the proband and her family, who carried HCM, showed relatively severe cardiac hypertrophy, with a high incidence of sudden death in females.

In family C, an Arg92Trp mutation was also found. The proband and her family, who carried HCM, showed mild cardiac hypertrophy and asymmetric septal hypertrophy. The proband's myocardial hypertrophy had been gradually increasing, and her mother had a suspected shift to the dilated phase.

In family D, the proband carried the new mutation Ile79Thr of *TNNT2*. She survived following an episode of Vf, and her cousin died suddenly at the age of 13 years. Previously, a mutation of the 79 residue, the Ile79Asn mutation, had been reported [24, 25], and this mutation also showed a poor prognosis. In our mutated case, even if the amino acid mutation (Ile79Thr) was different from the reported case (Ile79Asn), the patient showed a malignant prognosis. In family D, a 5-base-pair (CTTCT) deletion/deletion (D/D) polymorphism in intron 3 of the *TNNT2* was also found. It has been reported that this polymorphism had caused skipping of exon 4 of *TNNT2*, and that the deletion allele could be associated with a predisposition for prominent LVH [42]. Although it was in a very limited range, from our genetic study on this polymorphism we gained the impression that a patient carrying the *TNNT2* deletion/deletion polymorphism had a stronger tendency toward hypertrophy. To conduct further analysis, further examination including new cases is required.

We considered that, at least in Japanese familial HCM, only the type of genetic mutation of *TNNT2* did not seem useful in distinguishing the prognosis. However, if mutations were found, there was a risk of sudden death in youth. Therefore, regardless of the type of genetic mutation, it would be more important to observe the patient in detail from birth in each lineage.

The development of a “case-based” method would be useful to treat and help each individual, and more attention needs to be paid toward searching for the modifier and environmental factors including diet, lifestyle, exercise, and the modification gene and polymorphism.

Acknowledgments This work was supported by the Encouraging Development of Strategic Research Centers, Special Coordination Funds for Promoting Science and Technology, Ministry of Education, Culture, Sports, Science, and Technology, Japan. We would like to thank Michiko Furutani and Yoshiyuki Furutani for preparation of the manuscript.

Open Access This article is distributed under the terms of the Creative Commons Attribution License which permits any use, distribution, and reproduction in any medium, provided the original author(s) and the source are credited.

References

1. Cirino AL, Ho C (2009) Familial hypertrophic cardiomyopathy overview. *Gene Rev.* <http://www.ncbi.nlm.nih.gov/books/NBK1768/>

2. Maron BJ (1997) Hypertrophic cardiomyopathy. *Lancet* 350:127–133
3. Seidman JG, Sideman C (2001) The genetic basis for cardiomyopathy: from mutation identification to mechanistic paradigms. *Cell* 104:557–567
4. Spirito P, Sideman CE, McKenna WJ, Maron BJ (1997) The management of hypertrophic cardiomyopathy. *N Engl J Med* 1336:775–785
5. Marian AJ, Roberts R (2001) The molecular genetic bases for hypertrophic cardiomyopathy. *J Mol Cell Cardiol* 33:655–670
6. Nakashima K, Kusakawa I, Yamamoto T, Hirabayashi S, Hosoya R, Shimizu W, Sumitomo N (2013) A left ventricular noncompaction in a patient with long QT syndrome caused by a *KCNQ1* mutation: a case report. *Heart Vessels* 28:126–129
7. Erdmann J, Daehmlow S, Wischke S, Senyuva M, Werner U, Raible J, Tanis N, Dyachenko S, Hummel M, Hetzer R, Regitz-Zagrosek V (2003) Mutation spectrum in a large cohort of unrelated consecutive patients with hypertrophic cardiomyopathy. *Clin Genet* 64(4):339–349
8. Forissier JF, Carrier L, Farza H, Bonne G, Bercovici J, Richard P, Hainque B, Townsend PJ, Yacoub MH, Faure S, Dubourg O, Millaire A, Hagege AA, Desnos M, Komajda M, Schwartz K (1996) Codon 102 of the cardiac troponin T gene is a putative hot spot for mutations in familial hypertrophic cardiomyopathy. *Circulation* 94:3069–3073
9. Fujino N, Shimizu M, Ino H, Yamaguchi M, Yasuda T, Nagata M, Konno T, Mabuchi H (2002) A novel mutation Lys273Glu in the cardiac Troponin T gene shows high degree of penetrance and transition from hypertrophic to dilated cardiomyopathy. *Am J Cardiol* 89:29–33
10. Niimura H, Patton KK, McKenna WJ, Soultis J, Maron BJ, Seidman JG, Seidman CE (2002) Sarcomere protein gene mutations in hypertrophic cardiomyopathy of the elderly. *Circulation* 105:446–451
11. Matsushita Y, Furukawa T, Kasanuki H, Nishibatake M, Kurihara Y, Ikeda A, Kamatani N, Takeshima H, Matsuoka R (2007) Mutation of junctophilin type 2 associated with hypertrophic cardiomyopathy. *J Hum Genet* 52:543–548
12. de Tombe PP (2003) Cardiac myofilaments: mechanics and regulation. *J Biomech* 36:721–730
13. Harada K, Potter JD (2004) Familial hypertrophic cardiomyopathy mutations from different functional regions of Troponin T result in different effects on the pH and Ca²⁺ sensitivity of cardiac muscle contraction. *J Biol Chem* 279(15):14488–14495
14. Lassalle MW (2010) Defective dynamic properties of human cardiac troponin T mutations. *Biosci Biotechnol Biochem* 74(1):82–91
15. Palm T, Graboski S, Hitchcock-DeGregori SE, Greenfield NJ (2001) Disease-causing mutations in cardiac Troponin T: identification of a critical tropomyosin-binding region. *Biophys J* 81:2827–2837
16. Redwood CS, Moolman-Smook JC, Watkins H (1999) Properties of mutant contractile proteins that cause hypertrophic cardiomyopathy. *Cardiovasc Res* 44:20–36
17. Uchino T, Isomoto S, Noguchi T, Ono K (2013) Window current through the T-type Ca²⁺ channel triggers the mechanism for cellular apoptosis via mitochondrial pathways. *Heart Vessels*. doi:10.1007/s00380-012-0316-8
18. Gomes AV, Barnes JA, Harada K, Potter JD (2004) Role of troponin T in disease. *Mol Cell Biochem* 263:115–129
19. Anan R, Shono H, Kisanuki A, Arima S, Nakano S, Tanaka H (1998) Patients with familial hypertrophic cardiomyopathy caused by a Phe110Ile missense mutation in the cardiac troponin T gene have variable cardiac morphologies and a favorable prognosis. *Circulation* 98:391–397
20. Nakaura H, Yanaga F, Ohtsuki I, Morimoto S (1999) Effects of missense mutations Phe110Ile and Glu244Asp in human cardiac troponin T on force generation in skinned cardiac muscle fibers. *J Biochem* 126(3):457–460
21. Song L, Zou Y, Wang J, Zhen Y, Lou K, Zhang Q, Wang X, Wang H, Li J, Hui R (2005) Mutations profile in Chinese patients with hypertrophic cardiomyopathy. *Clin Chim Acta* 351:209–216
22. Moolman-Smook JC, De Lange WJ, Bruwer ECD, Brink PA, Corfield VA (1999) The origins of hypertrophic cardiomyopathy-causing mutations in two South African subpopulations: a unique profile of both independent and founder events. *Am J Hum Genet* 65(5):1308–1320
23. Moolman JC, Corfield VA, Posen B, Ngumbela K, Watokins H (1997) Sudden death due to troponin T mutations. *J Am Coll Cardiol* 29:549–555
24. Thierfelder L, Watkins H, MacRae C, Lamas R, McKenna W, Vosberg HP, Seidman CE (1994) α -Tropomyosin and cardiac troponin T mutations cause familial hypertrophic cardiomyopathy: a disease of the sarcomere. *Cell* 77:701–712
25. Watkins H, McKenna W, Thierfelder L, Suk HJ, Anan R, O'Donogue A, Spirito P, Matsumori A, Moravec CE, Seidman JG (1995) Mutations in the genes for cardiac troponin T and α -tropomyosin in hypertrophic cardiomyopathy. *N Engl J Med* 332:1058–1064
26. Xu Q, Dewey S, Nguyen S, Gomes AD (2010) Malignant and benign mutations in familial cardiomyopathies: insights into mutations linked to complex cardiovascular phenotypes. *J Mol Cell Cardiol* 48:899–909
27. Mörner S, Richard P, Kazzam E, Hellman U, Hainque B, Schwartz K, Waldenström A (2003) Identification of the genotypes causing hypertrophic cardiomyopathy in northern Sweden. *J Mol Cell Cardiol* 35(7):841–849
28. Vamava A, Baboonian C, Davison F, de Cruz, Elliot PM (1999) A new mutation of the cardiac troponin T gene causing familial hypertrophic cardiomyopathy without left ventricular hypertrophy. *Heart* 82(5):621–624
29. Nakajima-Taniguchi C, Matsui H, Fujio Y, Nagata S, Kishimoto T, Yamauchi-Takahara K (1997) Novel missense mutation in cardiac troponin T gene found in Japanese patient with hypertrophic cardiomyopathy. *J Mol Cell Cardiol* 29:839–843
30. Ho CY, Lever HM, DeSanctis R, Farver CF, Seidman JG, Seidman CE (2000) Homozygous mutation in cardiac troponin T: implications for hypertrophic cardiomyopathy. *Circulation* 102(16):1950–1955
31. Hen Y, Iguchi N, Machida H, Takada K, Utanohara Y (2012) High signal intensity on T2-weighted cardiac magnetic resonance imaging correlates with the ventricular tachyarrhythmia in hypertrophic cardiomyopathy. *Heart Vessels*. doi:10.1007/s00380-012-0300-3
32. Landstrom Andrew P, Ho Carolyn Y, Ackerman Michael J (2010) Mutation type is not clinically useful in predicting prognosis in hypertrophic cardiomyopathy. *Circulation* 2010(122):2441–2450
33. Yoshida MC, Satoh H, Sasaki M, Semba K, Yamamoto T, Toyoshima K (1986) Regional location of novel yes-related proto-oncogene, syn, on human chromosome 6 at band q21. *Jpn J Cancer Res* 77:1059–1061
34. Berg JN, Gallione CJ, Stenzel TT, Johnson DW, Allen WP, Schwartz CE, Jackson CE, Porteous M, Marchuket D (1997) The activin receptor-like kinase 1 gene: genomic structure and mutations in hereditary hemorrhagic telangiectasia type 2. *Am J Hum Genet* 61:60–67
35. Deng Z, Morse JH, Slager SL, Cuervo N, Moore KJ, Venetos G, Kalachikov S, Cayanis E, Fischer SG, Barst RJ, Hodge SE, Knowles JA (2000) Familial primary pulmonary hypertension

- (gene PPH1) is caused by mutations in the bone morphogenetic protein receptor-II gene. *Am J Hum Genet* 67:737–744
36. Tanus-Santos JE, Desai M, Flockhart DA (2001) Effects of ethnicity on the distribution of clinically relevant endothelial nitric oxide variants. *Pharmacogenetics* 11:719–725
 37. Rigat B, Hubert C, Corvol P, Soubrier F (1992) PCR detection of the insertion/deletion polymorphism of the human angiotensin converting enzyme gene (DCP1) (dipeptidyl carboxypeptidase 1). *Nucleic Acids Res* 20(6):1433
 38. Van Driest SL, Ommen SR, Tajik AJ, Gersh BJ, Ackerman MJ (2005) Yield of genetic testing in hypertrophic cardiomyopathy. *Mayo Clin Proc* 80:739–744
 39. Van Driest SV, Ackerman MJ, Ommen SR, Shakur R, Will ML, Nishimura RA, Tajik AJ, Gersh BJ (2002) Prevalence and severity of “benign” mutations in the beta myosin heavy chain, cardiac troponin-T, and alpha tropomyosin genes in hypertrophic cardiomyopathy. *Circulation* 106:3085–3090
 40. Ackerman MJ, Van Driest SV, Ommen SR, Will ML, Nishimura RA, Tajik AJ, Gersh BJ (2002) Prevalence and age-dependence of malignant mutations in the beta-myosin heavy chain and troponin T gene in hypertrophic cardiomyopathy: a comprehensive outpatient perspective. *J Am Coll Cardiol* 39:2042–2048
 41. Menon S, Michels V, Pellikka P, Ballew J, Karst M, Herron K, Nelson S, Rodeheffer R, Olson T (2008) Cardiac troponin T mutation in familial cardiomyopathy with variable remodeling and restrictive physiology. *Clin Genet* 74:445–454
 42. Komamura K, Iwai N, Kokame K, Yasumura Y, Kim J, Yamagishi M, Tomoike H, Kitakaze M, Miyatake K (2004) The role of a common TNNT2 polymorphism in cardiac hypertrophy. *J Hum Genet* 49:129–133

“Pediatric Heart Component” Qualifying Criteria

<p>“Pediatric Primary Heart Surgeon”</p>	<ul style="list-style-type: none"> • Meet current Bylaws requirements for “Primary Heart Surgeon”, and • 8 heart transplants in patients younger than 18 years of age • 4 heart transplants in patients younger than 12 years of age • American Board of Thoracic Surgery Congenital Heart Certification
<p>“Pediatric Primary Heart Physician”</p>	<ul style="list-style-type: none"> • Meet current Bylaws requirements for “Primary Heart Physician”, and • Care for 8 heart transplant patients younger than 18 years of age • Care for 4 heart transplant patients younger than 12 years of age • American Board of Pediatrics: Sub-board of Pediatric Cardiology Certification

OPTN



“Pediatric Lung Component” Qualifying Criteria

<p>“Pediatric Primary Lung Surgeon”</p>	<ul style="list-style-type: none"> • Meet current Bylaws requirements for “Primary Lung Surgeon” • 6 lung transplants in patients younger than 18 years of age <p>OR</p> <ul style="list-style-type: none"> • American Board of Thoracic Surgery Congenital Heart Certification
<p>“Pediatric Primary Lung Physician”</p>	<ul style="list-style-type: none"> • Meet current Bylaws requirements for “Primary Lung Physician” • American Board of Pediatrics certification in pulmonology medicine possessed by the individual who meets the current Bylaws requirements OR another member of the lung transplant team.

OPTN



Pediatric Training and Experience Considerations in the Bylaws – Additional Considerations

- Similar to the current Bylaws pathways, these pediatric-specific requirements would be expected of the clinician over 2-5 years
- Alternative pathway for individuals who do not meet all of the pediatric-specific requirements, yet very experienced
- One individual could fill both, for example, the primary surgeon and the pediatric primary surgeon roles, but it doesn't need to be the same person.

OPTN



Questions?

- Heung Bae Kim, M.D., Committee Chair
heung.kim@childrens.harvard.edu
- Region # Representative
name@email
- Chad Waller, Committee Liaison
chad.waller@unos.org

OPTN



OPTN/UNOS Pediatric Transplantation Committee
Interim Report for
January 15, 2014
Teleconference

The Pediatric Transplantation Committee's (the Committee's) update at the OPTN/UNOS fall regional meetings focused on its recommendations for pediatric training and experience considerations to be included in the OPTN Bylaws. The Committee met via teleconference on January 15, 2014, to consider the feedback received at the fall regional meetings and potential changes to its recommendations.

The call began with Committee members and UNOS staff recapping questions and comments provided at each regional meeting. With the exception of two regions that didn't express much concern, similar general themes were raised (to varying degrees) at each of the regional meetings. The following issues were recognized as the primary themes of concern:

- Lack of supporting data.
- Recommended requirements will negatively impact pediatric transplant candidates' access.
- Recommended case volumes are too high.
- Transplanting adolescents/larger pediatric patients should not require an approved "pediatric component."

Prior to the call, Committee leadership and UNOS staff discussed possible modifications to the Committee's "pediatric component" key personnel recommendations to accommodate these concerns. After reviewing the regional meeting feedback, the Committee Chair began presenting possible ideas to modify the Committee's recommendations. The first presented idea would eliminate the requirement that the "pediatric key personnel" case volumes are met over a five year period. Including a broad, undefined timeframe to attain the set level of experience would necessitate the addition of to-be-determined requirements that reflect "currency" with pediatric transplantation; e.g., a percentage of the reported cases must have been performed in the past two years. This possible modification is intended to address concerns that the recommended case volumes are too high, and the potential for these Bylaws to impact pediatric candidates' access negatively. Committee members expressed concerns about vast time differences between when one attained their pediatric transplant training/experience and when they may be approved as a "pediatric primary surgeon" or "pediatric primary physician." Committee leadership pointed out that there are no current requirements to check or limit very infrequent involvement with difficult pediatric cases. Additionally, although seemingly intuitive, there is no definitive evidence that infrequently performing these procedures is necessarily problematic. As indicated by the regional meeting feedback, more stringent recommendations will bring more opposition, which likely means the proposal will not pass. The Chair reminded the Committee that it must keep these things in mind and search for an acceptable balance as it develops a proposal.

The Committee proceeded to discuss the criticism that its recommendations, and the need for such, lack supporting evidence. Early discussions indicated consensus that requirements need specific numbers of cases so that transplant hospitals are clear on what is required and so the Membership and Professional Standards Committee (MPSC) has clear parameters to evaluate “pediatric program” applications. Committee members reiterated that it is impossible in this situation to find numerical data that distinguish a significant difference for a requirement that reflects the minimum amount of training and experience. For example, the Committee cannot prove that eight pediatric heart transplants is superior to six (or 10) pediatric heart transplants as a minimum level of necessary experience. The Committee does not believe that its recommendations necessarily reflect profound expertise with pediatric transplants; rather, the requirements reflect that the individual has been exposed to pediatric transplantation and the unique considerations that these cases require. Committee members also pointed out the arbitrary case volumes in the current key personnel Bylaws that are routinely accepted today. The Committee did incorporate some logic in its recommendations by making them a consistent percentage (40%) of the current Bylaws’ case volume requirements for each organ-specific program.

The Committee also discussed if its recommendations should solely focus on the youngest/smallest pediatric patients. This would have the potential to address concerns that caring for adolescent patients should not require an approved “pediatric component.” The Committee generally agreed that excluding adolescent patients neglects crucial, unique matters faced by teenaged transplant patients (psycho-social matters, development issues, etc.) that require special pediatric expertise. The Committee also expressed concerns about defining “pediatric” other than the commonly accepted less than 18 years of age. A Committee member suggested another option of having pediatric program “tiers” to focus on different age groups (or weights) of patients less than 18 years old (e.g., less than 18-13 years, 12-6 years, 5-0 years).

The mention of patient size prompted the Committee to discuss possible Bylaws requirements that consider experience relative to patients’ weights and not their ages. Committee members referenced off-line conversations with transplant surgeons that revealed a general acceptance of the quantifiable risks associated with transplanting smaller children, and that transplanting these patients requires unique skills. Focusing on the patients’ weights (especially the smallest patients) is reasonable, but those cutoffs could also be criticized as arbitrary. It would be difficult to explain a difference between transplanting a kidney into a 20kg child as compared to a 19kg child.

Conversation returned to possible requirements to demonstrate currency. The Committee first considered the “primary pediatric kidney surgeon” requirements, and a suggestion that half of the pediatric transplants must have been performed over the last five years. Applying this logic to the Committee’s recommendations would require a “pediatric primary surgeon” to have completed six kidney transplants in patients younger than 18 years of age in the past five years. Committee members responded with the suggestion of five transplants in patients younger than 18 years of age in the past five years, simplifying the “currency” requirement to an average of one pediatric transplant per year over the past five years.

To evaluate how reasonable this requirement may be, the Committee referenced a data analysis UNOS staff prepared for the call that evaluated the total number of pediatric transplants performed at each program over the past five years (Figures 1-4). Although potential Bylaws will require “pediatric transplant program key personnel” to have a set level of training/experience, not the transplant program, this analysis provides an approximation of which programs may have difficulty meeting any potential requirements. The Chair asked the Committee to consider, as it reviews the data, the possibility of eliminating the recommendations focused on younger/smaller pediatric transplant recipients. This approach would rely on appropriate medical judgment and outcome reviews to influence which programs care for the youngest, most complicated pediatric cases. Proceeding in this fashion may be more supported by the community as it would avoid limiting high volume, successful programs that focus on older pediatric patients.

The Committee first reviewed an assessment of kidney programs that performed at least one pediatric transplant and whether the *program volumes* could meet the Committee’s current recommendations

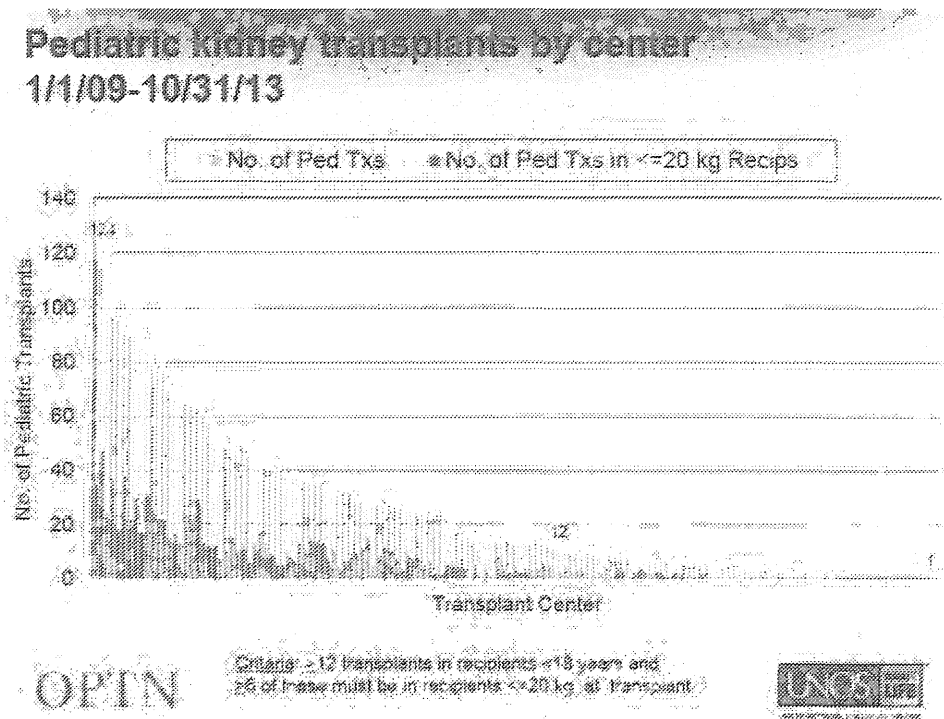


Figure 1: Pediatric kidney transplants by center, 1/1/09- 10/31/13

Figure 1 shows that 150 transplant programs performed at least one pediatric kidney transplant over the five year cohort examined (1/1/09-10/31/13). Of those 150 programs, 83 performed 12 or more pediatric kidney transplants, and 56 of those performed six or more pediatric transplants in recipients that weighed 20 kg or less at transplant. The Committee noted variability across transplant programs, highlighting that programs doing the most kidney transplants in patients that weigh 20 kg or less aren’t necessarily the same programs that do the most pediatric patients. This seems to support the notion that appropriate medical judgment and

outcome reviews will influence which programs care for the youngest, most complicated pediatric cases. Accordingly, the Committee was asked if it would be appropriate to remove the requirement that a “pediatric primary kidney surgeon” must have performed at least 6 transplants in patients that weigh 20 kg or less. Committee members asked for clarification on how outcomes are evaluated for programs that do occasional pediatric transplants. The Chair, who is also a member of the MPSC, responded that the MPSC evaluates a program’s pediatric and adult transplant outcomes separately. When evaluating a low-volume (currently defined as less than 10 transplants in two and a half years) of pediatric (or adult) outcomes, a single poor outcome flags that program for further review. Even though programs doing occasional pediatric transplants would have their outcomes monitored, some Committee members were still concerned that this would put the youngest/smallest transplant candidates in jeopardy of receiving substandard care. It is accepted that transplanting younger/smaller patients is more challenging, and mistakes in caring for these patients have particularly significant ramifications. In response, the Committee leadership reminded the Committee that this topic has been discussed multiple times over the years, and no solutions have been adopted because consensus has not been reached. The current Bylaws, or even if this Committee’s first recommendations were implemented immediately, do not prevent an individual with no pediatric transplant experience from transplanting a kidney into a 15 kg recipient. The Committee must remember that the recommended Bylaws will only address needed requirements for a “primary pediatric surgeon” and a “primary pediatric physician;” the expertise and experience of the program’s additional physicians and surgeons will not be addressed by the OPTN. An agreement on a basic pediatric program framework, thereby designating and acknowledging pediatric experience at programs that do pediatric transplants, would drastically improve the current Bylaws, even if it is not the Committee’s perfect solution. The Committee agreed to continue contemplating this possibility, and proceeded to review the remaining organ-specific data.

Pediatric liver transplants by center 1/1/09-10/31/13

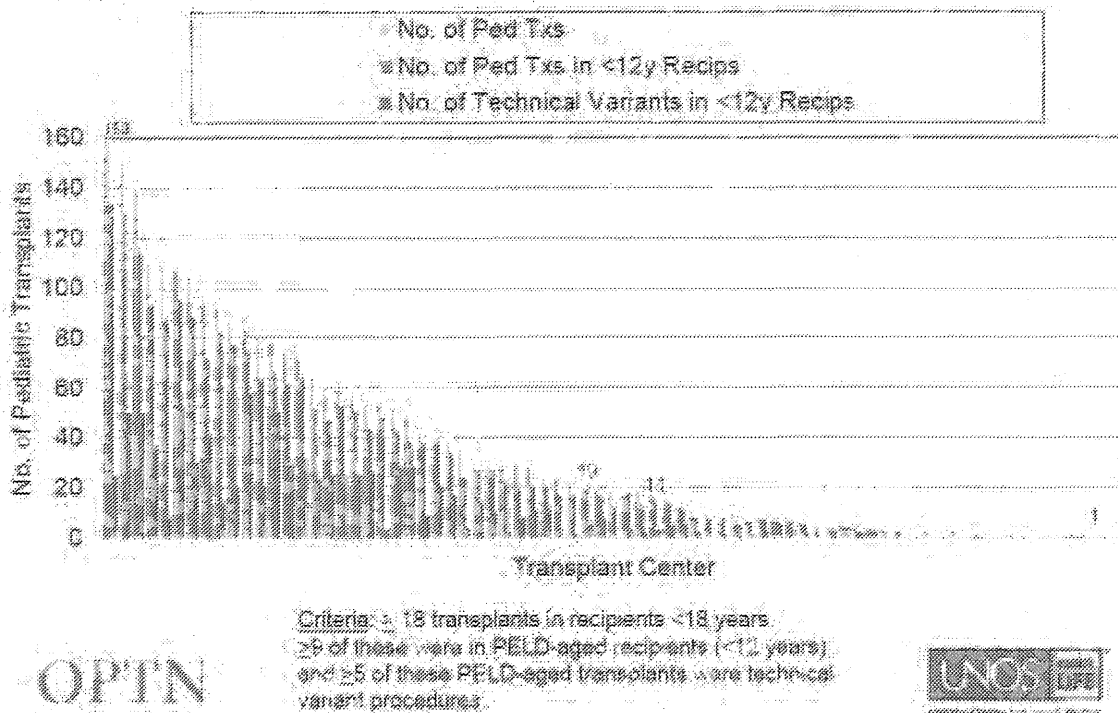


Figure 2: Pediatric liver transplants by center, 1/1/09- 10/31/13

Using the same cohort, the data displayed in Figure 2 show that 75 programs performed at least one pediatric liver transplant. Of these programs, 40 performed 18 or more pediatric liver transplants, 43 programs performed nine or more transplants in patients younger than 12 years old, and 36 programs performed five or more technical variant liver transplants in patients younger than 12 years old. This analysis yielded 32 transplant programs that met all three criteria. Again, the Committee noted program variation. The busiest pediatric liver program did relatively few technical variant transplants. In general, the technical variant requirement seems like it may be the most limiting. With these data, the Committee suggested that the technical variant requirement could inappropriately penalize those programs that are exposed to a high number of pediatric donors, and thus do not need to perform as many technical variant procedures. The Committee also highlighted that a program's number of liver transplants done in patients less than 12 years old is closely related to the program's total number of pediatric liver transplants. Committee members explained this observation by the fact that a majority of liver transplants are performed in pediatric patients younger than five years of age. Considering this parallel and the number of programs that may have difficulty qualifying with the technical variant requirement, should the Committee eliminate the requirements that a "pediatric primary liver surgeons" must perform nine or more transplants in patients less than 12, at least five of which must be technical variant procedures? Considering the close relationship, the Committee also considered if it should recommend only a number of transplants in patients younger than 12 years. Ultimately, participants agreed that the requirement should only focus on the number

of transplants performed in patients younger than 18 years. This would avoid potentially redefining “pediatric” and would accommodate those programs that occasionally transplant livers into teenagers.

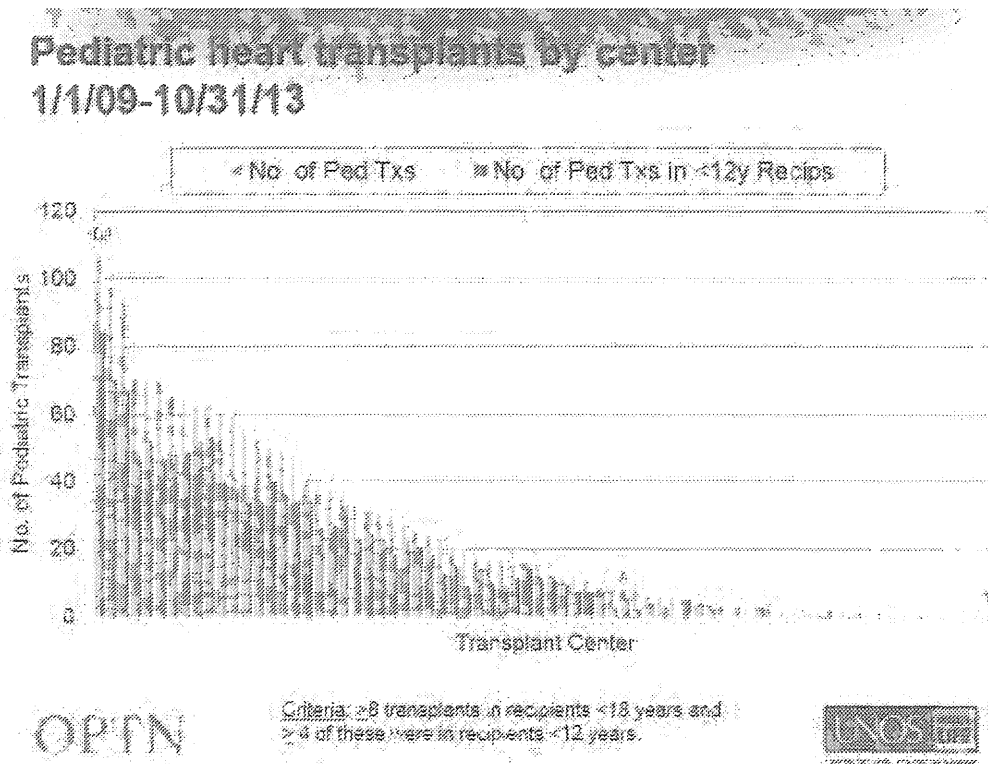
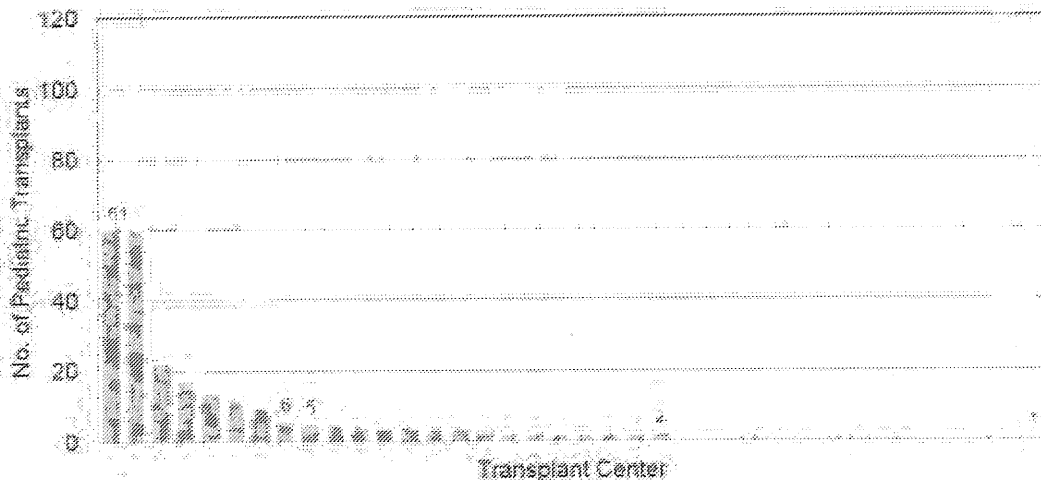


Figure 3: Pediatric heart transplants by center, 1/1/09- 10/31/13

Using the same cohort, the data show 74 programs performed at least one pediatric heart transplant (Figure 3). Of those, 45 programs performed 8 or more pediatric heart transplants and 46 programs performed four or more transplants in patients younger than 12. This analysis showed a total of 43 heart programs that meet the Committee’s recommended *case volume* requirements for a “primary pediatric heart surgeon.” Similar to liver, a significant number of pediatric heart transplants are performed in the youngest/smallest pediatric patients. As such, a program’s volume of heart transplants in patients younger than 12 years seems to be very closely related to the total number of pediatric heart transplants performed. To simplify, and address the concerns of those programs that want to transplant adolescents and not get involved with the youngest/smallest pediatric patients, it seems reasonable to remove all proposed requirements for “pediatric heart programs” with the exception of requiring eight or more pediatric heart transplants. This approach also addresses some concerns that were expressed regarding the inclusion of specific certifications.

**Pediatric lung transplants by center
1/1/09-10/31/13**



OPTN

Criteria: >6 transplants in recipients <18 years



Figure 4: Pediatric lung transplants by center, 1/1/09- 10/31/13

Using the same cohort, a total of 38 centers performed at least one pediatric lung transplant, eight of which did six or more pediatric transplants. The Committee thought that if the certification requirements would be removed for “pediatric primary heart surgeon,” then similar certification requirements should be removed for the “pediatric primary lung surgeon.” The Committee’s pulmonologist was unable to join the call, and the Chair agreed to reach out to him for additional input on the “pediatric lung program” requirements.

To conclude the call, the Committee agreed that Committee leadership and UNOS staff will draft a new set of recommendations based on the discussions had during this call. The new draft will be distributed to the Committee and another teleconference will be scheduled to discuss and refine these recommendations.

Participants:

Pediatric Transplantation Committee

Heung Bae Kim, MD (Committee Chair)

Eileen Brewer, MD (Vice-Chair)

Linda Addonizio, MD

Cecile Aguayo, BSN, RN, CCTC

Sandi Amaral, MD

Srinath Chinnakotla, MD, MCh

Dev Desai, MD, PhD

Michael Gautreaux, PhD, D.ABHI

Kenneth Lieberman, MD

Bret Mettler, MD

Greg Tiao, MD

HRSA

Ba Lin, MS, MPH

UNOS

Sally Aungier

Wida Cherikh, PhD

Betsy Gans

Jory Parker

Chad Waller, MS

Trevi Wilson, RN

SRTR

Susan Leppke, MPH

Jodi Smith, MD

SRTR SCIENTIFIC REGISTRY OF TRANSPLANT RECIPIENTS

[About the SRTR](#) | [Annual Data Reports](#) | [Transplant Program Reports](#) | [OPO Reports](#) | [For Researchers](#)

[Home](#) > [Program + Hospital Data](#) > [Find a Transplant Center](#) > [Compare Transplant Centers](#)

Compare US Hospitals with Heart Transplant Centers

WICH, WACH, VAUV, UTPC, TXTC, TNVU, TXCM, SCMU, PACH, PACP, OHCM, NYMS, OHCC, NYMA, NYCP, NCCM, NCMH, NCDU, MSUM, MOCG, MOCH, MNUM, MNSM, MIUM, MICH, MDJH, LAOF, MACH, ILUC, INIM, ILCM, FLUF, GAEH, FLJD, DEAI, FLAC, COCH, CAUC, AZSJ, AZCH, CACL, CAPC, CALL, ARCH, ALUA

PSR Quick Links

- [Transplant Program Reports](#)
- [Methodology](#)
- [Risk-Adjustment Models \(Transplant Programs\)](#)
- [Risk-Adjustment Models \(OPO\)](#)
- [Transplant Report Timeline](#)
- [OPO Report Timeline](#)
- [Past Notices](#)
- [FAQs](#)

Contact the SRTR

914 South 8th Street
Suite S-4.100
Minneapolis, MN
55404
Tel: (877) 970-SRTR
Fax: (612) 347-5878
[Email Us](#)

State	Hospital	As of 12/31/2011 Number of Candidates	Transplant (1/1/11 - 12/31/11)				After Transplant (1/1/09 - 6/30/11)		View Report
			Donor Type		Recipient Age		Patient Survival Rate - One Year		
			Living	Deceased	18 & Over	Under 18	18 & Over	Under 18	
OH	Children's Hospital Medical Center, Cincinnati, OH	12		9	0	9	Not Applicable	100% 	New Format Traditional Format
PA	Children's Hospital of Pittsburgh of UPMC, Pittsburgh, PA	4		9	1	8	100% 	100% 	New Format Traditional Format
PA	Children's Hospital of Philadelphia, Philadelphia, PA	13		10	0	10	100% 	93% 	New Format Traditional Format
SC	Medical University of South Carolina, Charleston, SC	15		24	19	5	100% 	80% 	New Format Traditional Format
TN	Vanderbilt University Medical Center, Nashville, TN	38		36	29	7	100% 	96% 	New Format Traditional Format
TX	Children's Medical Center of Dallas, Dallas, TX	30		9	0	9	100% 	89% 	New Format Traditional Format
TX	Texas Children's Hospital, Houston, TX	21		15	5	10	100% 	97% 	New Format Traditional Format
UT	Primary Children's Medical Center, Salt Lake City, UT	4		6	0	6	100% 	88% 	New Format Traditional Format
VA	University of Virginia Health Sciences Center, Charlottesville, VA	20		15	13	2	82% 	100% 	New Format Traditional Format
WA	Children's Hospital & Regional Medical Center, Seattle, WA	8		10	1	9	Not Applicable	92% 	New Format Traditional Format
WI	Children's Hospital of Wisconsin, Milwaukee, WI	19		13	0	13	100% 	91% 	New Format Traditional Format

The SRTR is administered by the Chronic Disease Research Group of the Minneapolis Medical Research Foundation, with oversight and funding from the Health Resources and Services Administration.

[Citation Advice](#)

[HRSA](#) | [HHS](#) | [Viewers & Players](#) | [Freedom of Information Act](#) | [Privacy Policy](#) | [Disclaimers](#) | [Accessibility](#)

SRTR SCIENTIFIC REGISTRY OF TRANSPLANT RECIPIENTS

[About the SRTR](#) | [Annual Data Reports](#) | [Transplant Program Reports](#) | [OPO Reports](#) | [For Researchers](#)

[Home](#) > [Program + Hospital Data](#) > [Find a Transplant Center](#) > [Compare Transplant Centers](#)

Compare US Hospitals with Heart Transplant Centers

NYMS, OHCC, NYMA, NYCP, NCCM, NCMH, NCDU, MSUM, MOCG, MOCH, MNUM, MNMS, MIUM, MICH, MDJH, LAOF, MACH, ILUC, INIM, ILCM, FLUF, GAEH, FLJD, DEAI, FLAC, COCH, CAUC, AZSJ, AZCH, CACL, CAPC, CALL, ARCH, ALUA

State	Hospital	As of 12/31/2011 Number of Candidates	Transplant (1/1/11 - 12/31/11)		Recipient Age		After Transplant (1/1/09 - 6/30/11)		View Report
			Donor Type		18 & Under		Patient Survival Rate - One Year		
			Living	Deceased	18 & Over	Under 18	18 & Over	Under 18	
MN	University of Minnesota Medical Center, Fairview, Minneapolis, MN	62		23	15	8	93%	73%	New Format Traditional Format
MO	Cardinal Glennon Children's Hospital, St. Louis, MO	0		2	0	2	Not Applicable	100%	New Format Traditional Format
MO	St. Louis Children's Hospital, St Louis, MO	16		18	0	18	100%	87%	New Format Traditional Format
MS	University of Mississippi Medical Center, Jackson, MS	23		13	10	3	76%	Not Applicable	New Format Traditional Format
NC	Carolinas Medical Center, Charlotte, NC	11		18	16	2	96%	100%	New Format Traditional Format
NC	Duke University Hospital, Durham, NC	65		60	52	8	93%	83%	New Format Traditional Format
NC	University of North Carolina Hospitals, Chapel Hill, NC	19		19	16	3	86%	100%	New Format Traditional Format
NY	NY Presbyterian Hospital/Columbia Univ. Medical Center, New York, NY	173		77	54	23	89%	91%	New Format Traditional Format
NY	Montefiore Medical Center, Bronx, NY	23		29	20	9	97%	100%	New Format Traditional Format
NY	Mount Sinai Medical Center, New York, NY	34		29	27	2	85%	70%	New Format Traditional Format
OH	The Cleveland Clinic Foundation, Cleveland, OH	101		55	53	2	96%	100%	New Format Traditional Format

PSR Quick Links

- [Transplant Program Reports](#)
- [Methodology](#)
- [Risk-Adjustment Models \(Transplant Programs\)](#)
- [Risk-Adjustment Models \(OPO\)](#)
- [Transplant Report Timeline](#)
- [OPO Report Timeline](#)
- [Past Notices](#)
- [FAQs](#)

Contact the SRTR

914 South 8th Street
Suite S-4.100
Minneapolis, MN
55404
Tel: (877) 970-SRTR
Fax: (612) 347-5878
[Email Us](#)

The SRTR is administered by the Chronic Disease Research Group of the Minneapolis Medical Research Foundation, with oversight and funding from the Health Resources and Services Administration.

[Citation Advice](#)

[HRSA](#) | [HHS](#) | [Viewers & Players](#) | [Freedom of Information Act](#) | [Privacy Policy](#) | [Disclaimers](#) | [Accessibility](#)

SRTR SCIENTIFIC REGISTRY OF TRANSPLANT RECIPIENTS

[About the SRTR](#) | [Annual Data Reports](#) | [Transplant Program Reports](#) | [OPO Reports](#) | [For Researchers](#)

[Home](#) > [Program + Hospital Data](#) > [Find a Transplant Center](#) > [Compare Transplant Centers](#)

Compare US Hospitals with Heart Transplant Centers

MNSM, MIUM, MICH, MDJH, LAOF, MACH, ILUC, INIM, ILCM, FLUF, GAEH, FLJD, DEAI, FLAC, COCH, CAUC, AZSJ, AZCH, CACL, CAPC, CALL, ARCH, ALUA

PSR Quick Links

- [Transplant Program Reports](#)
- [Methodology](#)
- [Risk-Adjustment Models \(Transplant Programs\)](#)
- [Risk-Adjustment Models \(OPO\)](#)
- [Transplant Report Timeline](#)
- [OPO Report Timeline](#)
- [Past Notices](#)
- [FAQs](#)

Contact the SRTR

914 South 8th Street
Suite S-4.100
Minneapolis, MN
55404
Tel: (877) 970-SRTR
Fax: (612) 347-5878
[Email Us](#)

State	Hospital	As of 12/31/2011 Number of Candidates	Transplant (1/1/11 - 12/31/11)				After Transplant (1/1/09 - 6/30/11)		View Report
			Donor Type		Recipient Age		Patient Survival Rate - One Year		
			Living	Deceased	18 & Over	Under 18	18 & Over	Under 18	
FL	Shands Hospital at The University of Florida, Gainesville, FL	59		31	20	11	93%	90%	New Format Traditional Format
GA	Children's Healthcare of Atlanta at Egleston, Atlanta, GA	2		17	1	16	Not Applicable	94%	New Format Traditional Format
IL	Children's Memorial Hospital, Chicago, IL	6		13	0	13	100%	90%	New Format Traditional Format
IL	University of Chicago Medical Center, Chicago, IL	41		25	23	2	83%	100%	New Format Traditional Format
IN	Indiana University Health, Indianapolis, IN	10		22	19	3	91%	100%	New Format Traditional Format
LA	Ochsner Foundation Hospital, New Orleans, LA	38		25	23	2	98%	100%	New Format Traditional Format
MA	Children's Hospital, Boston, MA	11		11	0	11	0%	96%	New Format Traditional Format
MD	Johns Hopkins Hospital, Baltimore, MD	36		16	10	6	76%	94%	New Format Traditional Format
MI	Children's Hospital of Michigan, Detroit, MI	3		3	1	2	100%	75%	New Format Traditional Format
MI	University of Michigan Medical Center, Ann Arbor, MI	50		33	29	4	89%	100%	New Format Traditional Format
MN	Saint Marys Hospital (Mayo Clinic), Rochester, MN	83		29	24	5	96%	100%	New Format Traditional Format

The SRTR is administered by the Chronic Disease Research Group of the Minneapolis Medical Research Foundation, with oversight and funding from the Health Resources and Services Administration.

[Citation Advice](#)

[HRSA](#) | [HHS](#) | [Viewers & Players](#) | [Freedom of Information Act](#) | [Privacy Policy](#) | [Disclaimers](#) | [Accessibility](#)

SRTR SCIENTIFIC REGISTRY OF TRANSPLANT RECIPIENTS

[About the SRTR](#) | [Annual Data Reports](#) | [Transplant Program Reports](#) | [OPO Reports](#) | [For Researchers](#)

[Home](#) > [Program + Hospital Data](#) > [Find a Transplant Center](#) > [Compare Transplant Centers](#)

Compare US Hospitals with Heart Transplant Centers

FLJD, DEAI, FLAC, COCH, CAUC, AZSJ, AZCH, CACL, CAPC, CALL, ARCH, ALUA

State	Hospital	As of 12/31/2011 Number of Candidates	Transplant (1/1/11 - 12/31/11)				After Transplant (1/1/09 - 6/30/11)		View Report
			Donor Type		Recipient Age		Patient Survival Rate - One Year		
			Living	Deceased	18 & Over	Under 18	18 & Over	Under 18	
AR	Arkansas Children's Hospital, Little Rock, AR	6		31	0	31	100% <small>AS EXPECTED</small>	84% <small>AS EXPECTED</small>	New Format Traditional Format
AZ	Phoenix Children's Hospital, Phoenix, AZ	2		2	0	2	Not Applicable	Not Applicable	New Format Traditional Format
AZ	St Josephs Hospital and Medical Center, Phoenix, AZ	0		4	0	4	Not Applicable	100% <small>AS EXPECTED</small>	New Format Traditional Format
CA	Childrens Hospital Los Angeles, Los Angeles, CA	4		3	0	3	Not Applicable	100% <small>AS EXPECTED</small>	New Format Traditional Format
CA	Loma Linda University Medical Center, Loma Linda, CA	14		24	10	14	94% <small>AS EXPECTED</small>	88% <small>AS EXPECTED</small>	New Format Traditional Format
CA	Lucile Salter Packard Childrens Hospital at Stanford, Palo Alto, CA	6		13	1	12	100% <small>AS EXPECTED</small>	90% <small>AS EXPECTED</small>	New Format Traditional Format
CA	University of California at Los Angeles Medical Center, Los Angeles, CA	66		52	35	17	94% <small>AS EXPECTED</small>	84% <small>AS EXPECTED</small>	New Format Traditional Format
CO	The Children's Hospital, Aurora, CO	4		16	2	14	100% <small>AS EXPECTED</small>	95% <small>AS EXPECTED</small>	New Format Traditional Format
DE	Alfred I DuPont Hospital for Children, Wilmington, DE	1		7	0	7	Not Applicable	100% <small>AS EXPECTED</small>	New Format Traditional Format
FL	All Children's Hospital, St. Petersburg, FL	0		6	0	6	Not Applicable	100% <small>AS EXPECTED</small>	New Format Traditional Format
FL	Joe DiMaggio Children's Hospital, Hollywood, FL	2		3	0	3	Not Applicable	100% <small>AS EXPECTED</small>	New Format Traditional Format

PSR Quick Links

- [Transplant Program Reports](#)
- [Methodology](#)
- [Risk-Adjustment Models \(Transplant Programs\)](#)
- [Risk-Adjustment Models \(OPO\)](#)
- [Transplant Report Timeline](#)
- [OPO Report Timeline](#)
- [Past Notices](#)
- [FAQs](#)

Contact the SRTR

914 South 8th Street
Suite S-4.100
Minneapolis, MN
55404
Tel: (877) 970-SRTR
Fax: (612) 347-5878
[Email Us](#)

The SRTR is administered by the Chronic Disease Research Group of the Minneapolis Medical Research Foundation, with oversight and funding from the Health Resources and Services Administration.

[Citation Advice](#)

[HRSA](#) | [HHS](#) | [Viewers & Players](#) | [Freedom of Information Act](#) | [Privacy Policy](#) | [Disclaimers](#) | [Accessibility](#)



[About the SRTR](#) | [Annual Data Reports](#) | [Transplant Program Reports](#) | [OPO Reports](#) | [For Researchers](#)

[Home](#) > [Program + Hospital Data](#) > [Find a Transplant Center](#) > [Compare Transplant Centers](#)

Compare US Hospitals with Heart Transplant Centers

WASH, VAMC, PRCC, OHCH, NEUN, KYKC, IAIV, ALUA

State	Hospital	As of 12/31/2011 Number of Candidates	Transplant (1/1/11 - 12/31/11)				After Transplant (1/1/09 - 6/30/11)		View Report
			Donor Type		Recipient Age		Patient Survival Rate - One Year		
			Living	Deceased	18 & Over	Under 18	18 & Over	Under 18	
AL	University of Alabama Hospital, Birmingham, AL	23		32	22	10	94% <small>AS EXPECTED</small>	100% <small>AS EXPECTED</small>	New Format Traditional Format
IA	University of Iowa Hospitals and Clinics, Iowa City, IA	13		16	15	1	85% <small>AS EXPECTED</small>	100% <small>AS EXPECTED</small>	New Format Traditional Format
KY	Kosair Childrens Hospital, Louisville, KY	0		1	0	1	Not Applicable	50% <small>AS EXPECTED</small>	New Format Traditional Format
NE	The Nebraska Medical Center, Omaha, NE	14		17	16	1	88% <small>AS EXPECTED</small>	100% <small>AS EXPECTED</small>	New Format Traditional Format
OH	Nationwide Children's Hospital, Columbus, OH	3		1	0	1	100% <small>AS EXPECTED</small>	86% <small>AS EXPECTED</small>	New Format Traditional Format
PR	Cardiovascular Center of Puerto Rico and the Caribbean, San Juan, PR	1		6	5	1	93% <small>AS EXPECTED</small>	100% <small>AS EXPECTED</small>	New Format Traditional Format
VA	Medical College of Virginia Hospitals, Richmond, VA	35		19	18	1	84% <small>AS EXPECTED</small>	Not Applicable	New Format Traditional Format
WA	Sacred Heart Medical Center, Spokane, WA	17		10	9	1	88% <small>AS EXPECTED</small>	Not Applicable	New Format Traditional Format

PSR Quick Links

- [Transplant Program Reports](#)
- [Methodology](#)
- [Risk-Adjustment Models \(Transplant Programs\)](#)
- [Risk-Adjustment Models \(OPO\)](#)
- [Transplant Report Timeline](#)
- [OPO Report Timeline](#)
- [Past Notices](#)
- [FAQs](#)

Contact the SRTR

914 South 8th Street
Suite S-4.100
Minneapolis, MN
55404
Tel: (877) 970-SRTR
Fax: (612) 347-5878
[Email Us](#)

The SRTR is administered by the Chronic Disease Research Group of the Minneapolis Medical Research Foundation, with oversight and funding from the Health Resources and Services Administration.

[Citation Advice](#)

[HRSA](#) | [HHS](#) | [Viewers & Players](#) | [Freedom of Information Act](#) | [Privacy Policy](#) | [Disclaimers](#) | [Accessibility](#)