

Production of tissue-engineered small intestine to restore normal intestinal function via autologous transplantation.

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Introduction

The surgical replacement of the small intestine for chronic and acute cases of intestinal failure is known as intestinal transplantation (intestinal transplantation, or small bowel transplantation). While other medicines such as parenteral nutrition (PN) can sometimes be used to treat intestinal failure, problems such as PN-associated liver disease and small bowel syndrome may make transplantation the only viable option. Intestinal transplantation is one of the most uncommon types of organ transplantation, however it is becoming more common as a therapeutic option because to advancements in immunosuppressive regimens, surgical technique, PN, and clinical treatment of pre- and post-transplant patients.

Pre-transplant diagnoses and short bowel syndrome

The inability to absorb nutrients, fluids, and electrolytes from food would be life-threatening if the small intestine failed. Homeostasis cannot be maintained without these vital components and the ability to maintain energy balances, and one's prognosis will be poor. Intestinal failure can be caused by a variety of factors, including nutritional, viral, traumatic, and metabolic problems that impair normal architecture and physiology. Many of the underlying problems that lead to failure are genetic or congenital in origin. Severe inflammation, ulceration, bowel blockage, fistulation, perforation, and other Crohn's disease pathologies, for example, can substantially impair intestinal function. Despite the dangers that these disorders may offer in and of themselves, they may lead to even more serious consequences that require the diseased intestine to be replaced. Short bowel syndrome, which is commonly a subsequent symptom of another intestinal disease, is the most common reason for an intestinal transplant. In 2008, short-bowel syndrome was the cause of 73% of intestinal transplants in the United States, followed by functional bowel issues (15%) and other causes (12%). Natural SBS is extremely rare, with an estimated prevalence of 3 per 100,000 births. The most common cause is surgical removal, which is done to address a variety of gastroenterological and congenital diseases include Crohn's disease, necrotizing enterocolitis, mesenteric ischemia, motility dysfunction, omphalocele/gastroschisis, tumours, and volvulus [1-3].

Alternative treatments

The loss of intestinal function, regardless of the underlying illness, does not always necessitate a transplant. Other

surgical and nonsurgical treatments may be sufficient to address many disorders, such as necrotizing enterocolitis or volvulus, especially if SBS does not develop. Through PN, an individual can absorb nutrients intravenously, avoiding food consumption and digestion totally. Long-term life with SBS and without PN is achievable with enteral nutrition, but for many patients, this is insufficient because it relies on the remaining intestine's ability to adapt and increase its absorptive capacity. Anyone can get PN, despite the fact that it is more difficult and expensive to execute. Although PN can cover all energy, hydration, and nutrient demands and may be done at home, it can have a major impact on quality of life. PN takes 10 to 16 hours on average to administer, but it can take up to 24 hours. Attachment to the IV pump might severely impede daily life during this time period. PN can cause a variety of health problems over time, including extreme dehydration, catheter-related infections, and liver disease. Up to 50% of individuals with PN-associated liver disease die within 5–7 years, with a mortality rate of 2–50 percent [4-6].

Surgical bowel lengthening using serial transverse enteroplasty (STEP) or the older longitudinal intestinal lengthening and tailoring (LILT) is an alternative to transplant for people with SBS. Although both treatments contribute to a 70% increase in lifespan, STEP looks to be slightly better in terms of mortality and transplant progression. Nonetheless, a positive response to either technique may minimise the amount of PN necessary, if not eliminate it entirely [7].

References

1. Skandalakis JE, Skandalakis LJ, Skandalakis PN. Small intestine. In: *Surgical Anatomy and Technique*. Springer, New York, NY, 2000.
2. Doherty MM, Charman WN. The mucosa of the small intestine. *Clin Pharmacokinet*. 2002;41(4):235-53.
3. Bayliss WM, Starling EH. The movements and innervation of the small intestine. *J Physiol*. 1899;24(2):99.
4. Schanker LS, Tocco DJ, Brodie BB, et al. Absorption of drugs from the rat small intestine. *J Pharmacol Exp Ther*. 1958;123(1):81-8.
5. Ouriel K, Adams JT. Adenocarcinoma of the small intestine. *Am J Surg*. 1984;147(1):66-71.

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6. King CE, Toskes PP. Small intestine bacterial overgrowth. *Gastroenterol.* 1979;76(5):1035-55.
7. Darling RC, Welch CE. Tumors of the small intestine. *NEJM.* 1959;260(9):397-408.