Dr. M. P. Scheid's name was misspelled in her article that appeared in the October 1982 issue. To ensure that her article is properly cited in the future, please replace page 1057 of volume 156 with this page.

T CELL DEVELOPMENT IN NORMAL AND THYMOPENTIN-TREATED NUDE MICE*

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There has been much uncertainty regarding the presence, variety, and maturity of T cells in athymic mice. Placental passage of maternal T cells cannot account for T cells in *nu/nu* progeny of *nu/nu* parents (1, and unpublished observations), and there is good evidence against the hypothesis of a functional thymic rudiment (2).

Prothymocytes, which are present equally in athymic and normal mice, are already committed to express the thymocyte surface phenotype and can be induced to do so by many nonspecific, as well as thymus-related, agents. Thus, nonspecific induction, attributable to incidental conditions, such as endemic viral hepatitis (3), which vary from one mouse colony to another, can explain some of the inconsistencies among published reports, at least as these concern surface phenotype.

As regards function, *nu/nu* splenocytes, cultured under special conditions including provision of interleukin 2 (IL-2)[1] (4), respond to T cell mitogens and allogeneic stimulator cells (mixed leukocyte culture) and can yield cloned lines of allogeneic killer effector cells (5). It has even been suggested that lack of IL-2 could be the only primary deficiency resulting from lack of a thymus (6).

In the present study, we sought to define the T cell population of *nu/nu* mice, specific pathogen free (SPF) or germ free, in more detail, according to the criteria of diversity of Ly sets and expression of the markers TL and Qa-1, and to see how the proportions of T cells expressing these phenotypes may be affected by treatment with thymopentin (TP-5), a synthetic pentapeptide fragment of thymopoietin.