speculate that genetics is playing a significant role in the determination of the development of a particular type of leprosy in a person.

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Lupus and Lepros

TO THE EDITOR:

For many centuries skin tuberculosis has been termed lupus vulgaris. The great German pathologist Rudolf Virchow ([6]) was intrigued by this name and established that it had appeared in the writings of the masters of the Salerno school of medicine, founded in the 10th century, and particularly in those of Roger of Salerno (ca. 1180). Nevertheless the origin of the term remained obscure.

It is generally assumed that the word *lupus* (Latin: a wolf) alludes to the tissue destruction characteristic of tuberculosis. In 1736, for example, Turner remarked that ". . . it is termed lupus, for that is, say some, of a ravenous nature, and like that fierce creature, not satisfy'd but with flesh" ([5]). Paradoxically, though, lupus vulgaris is an extremely chronic affliction: the very slow progression of the destructive process is in strong contrast to the feeding habits of even the most indolent of wolves.

Could lupus, therefore, be a corruption of some other word used to describe a chronic and disfiguring skin disease? An intriguing possibility is that it originated from the same Greek word, *lepros,* from which leprosy was derived. This word originally denominated various skin diseases characterized by peeling and was used to translate the Hebrew word *Tsara'ath.* (Leprosy, as we now define it, was known by the Greeks as *Elephantiasis Graecorum.*) This, in turn, raises the possibility that the lesions termed *Tsara'ath* in the old Testament and Gospels included skin tuberculosis. At a time when tuberculosis was prevalent in cattle in Great Britain, many cases of lupus vulgaris were seen and over half were due to bovine tubercle bacilli ([4]). There is ample evidence that cattle farming was well established in ancient Israel, and it has been suggested that the "wen" of cattle (Leviticus 22:22) referred to tuberculosis ([3]). (Pulmonary tuberculosis also afflicted the Israelites and was termed *Shachepheth.*) Thus, there is a strong likelihood that lupus vulgaris occurred in Israel before and during the time of Christ and that it was included in the conditions termed *Tsara'ath* and, subsequently, *lepros.* Hence the names for skin lesions due to *Mycobacterium leprae* and to *M. tuberculosis* could have a common etymological origin.

As *Tsara'ath* was amenable to healing by the laying on of hands (Luke 5:12–15), it has been suggested that the disease had a psycogenic rather than an organic cause ([2]). On the other hand, it is noteworthy that scrofula, lupus vulgaris, and other nonpulmonary manifestations of tuberculosis were, for many centuries, considered curable by the touch of a reigning monarch, hence the collective epithet "King's Evil" ([1, 7]). The belief that this gift was bestowed by Divine Grace, and Christ's particular directions to His followers that they should heal the victims of *Tsara'ath* (Matthew 10:8), established a further speculative link between Biblical "leprosy" and tuberculosis.

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Determination of the D and L Configuration of Phenolic Glycolipids of *M. leprae* and *M. bovis*

TO THE EDITOR:

It has been reported by Hunter, *et al.* that the structure of the sugar part of the species-specific phenolic glycolipid-I (PGL-I) of *Mycobacterium leprae* is *O*-(3,6-di-*O*-methyl-β-D-glucopyranosyl)-(1→4)-*O*-(2,3-di-*O*-methyl-α-L-rhamnopyranosyl)-(1→2)-3-*O*-methyl-α-L-rhamnopyranose ([15, 16]). This structure has generally been accepted and used elsewhere. However, the absolute structure of each sugar has only been assumed and has not been determined in that report ([16]). Several laboratories have shown by synthetic study that the disaccharide and trisaccharide of PGL-I synthesized from D-glucose and L-rhamnose have almost the same activity as that of PGL-I ([1–4, 6–10]). This suggests that the assumption of Hunter, *et al.* is correct, but there has been no direct evidence concerning the absolute configurations of the sugar residues. On the other hand, Demarteau-Ginsberg and Lederer have reported that the structure of the sugar part of the PGL of *M. bovis* (mycoside B) is 2-*O*-methyl-D-rhamnose, which was determined by an optical rotation study ([5]). However, D-rhamnose is a very rare sugar, and the reports which appeared after that paper did not treat the absolute configuration of the sugar residue ([11, 12]). Therefore, it is necessary to make sure of this determination by a more direct method. This paper provides the gas chromatographic determination of the absolute structure of the sugar residues of the PGLs of *M. leprae* and *M. bovis.*

Analytical procedures of the absolute configuration were based on the glycosidation with optically active alcohol, (+)-2-butanol ([13]). Five hundred μg of PGL-I from human-*M. leprae*-infected armadillo liver, synthesized trisaccharide, *p*-(2-methoxycarbonylethyl)phenyl *O*-(3,6-di-*O*-methyl-β-D-glucopyranosyl)-(1→4)-*O*-(2,3-di-*O*-methyl-α-L-rhamnopyranosyl)-(1→2)-3-*O*-methyl-α-L-rhamnopyranoside ([9]), or synthesized 2-*O*-methyl-L-rhamnopyranose was heated for 15 hr in 0.5 ml of (±)-2-butanol or (+)-2-butanol in the presence of 25 mg of powdered Amberlite IR 120 (H^+). The mixture was filtered, evaporated with the repeated addition of methanol, and then dissolved in 1 ml of methanol. Insoluble materials were filtered out with the aid of Celite. It was evaporated and dried. The residue was trimethylsilylated with 0.1 ml of TMS-PZ (Tokyo Kasei Co.) by heating the mixture at 40°C for 30 min, and an aliquot was analyzed by a capillary gas chromatograph (Hitachi G3000). Gas-liquid chromatography (GLC) conditions were as follows: Column; chemical bonded OV-1; d.f. 0.5 μm; length 5 m (0.25 mm i.d.); temp. program = 150°C for 3 min then →200°C at 4°C/min; carrier gas = 34 cm/min; split ratio = 25:1; injection temp. = 220°C.

Figure 1 shows the results of GLC of the

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FIG. 1. GLC of the sugar derivatives of the PGL-I of *M. leprae*: (+)-butanol-treated PGL-I (a), (±)-butanol-treated PGL-I (b), and (+)-butanol-treated synthetic trisaccharide (c) were trimethylsilylated and subjected to capillary GLC. (GLC conditions are given in the text.)

FIG. 2. GLC of the sugar derivatives of the phenolic glycolipid of *M. bovis*: (+)-butanol-treated PGL (a), (±)-butanol-treated PGL (b), and (+)-butanol-treated 2-*O*-methyl-L-rhamnopyranose (c) were trimethylsilylated and subjected to capillary GLC.