MATERNAL AGE IN FAMILIAL MONGOLISM.*

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IN recent years several writers have attempted to bring the known aetiological effect of maternal age in mongolism into direct relationship with its causation. Brousseau (1928) sifted the available data very carefully, but failed to come to any conclusion as to how maternal age could exert its effect. Benda (1947) considers that advanced age is only a subsidiary factor making maternal illness or endocrine disorder more significant, and a similar view seems to be held by Ingalls (1947). The conclusion is drawn by Geyer (1939) that mongolism is due to an abnormal ovum; if so, the older the mother, presumably the more likely are ova to be abnormal. Conversely, Engler (1949) believes that the foetal dysplasia is caused by faulty embedding of a normal ovum in uterine mucosa which has deteriorated in consequence of age, infection or surgical interference. Jenkins (1933) lays stress on the analogy with certain phenomena seen in animal genetics, for Wright (1926) had shown that the manifestation of hereditary polydactyly in guinea-pigs was influenced by the age of the dam: young mothers produced more polydactylous offspring than older ones. A similar effect has been found by Holt (1947) in polydactylous mice. There is no experimental evidence as to the nature of the mechanism involved, but it may be supposed to be physical or chemical. The search for a process akin to antigenic incompatibility has also been suggested (Penrose, 1946), immunity developing with greater ease in maturer than in younger maternal tissues. It is not necessary to look for a specifically pathological process, however, since the maternal-foetal reaction might be analogous to that in the aetiology of fraternal twins, which especially occurs at late maternal ages, which centre around 38 years.

Information about the relation of maternal age to the incidence of mongolism has been collected by innumerable observers, among the most useful surveys being those of van der Scheer (1927) and Orel (1927). Very little attention has been paid, however, to the peculiarities of maternal age which are noticeable in familial cases, that is, to the genetical investigation of maternal age effects themselves. To do this a great amount of material is required and, since the familial incidence of mongolism is slight, the accumulation of sufficient data to make analysis worth while takes many years. In the present paper all available family data from the general literature, in which maternal ages were given, is included. To this has been added a number of families investigated in connection with the writer's Colchester Survey (Penrose, 1938). Further instances were personally communicated by Halperin (1939) and

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Ford-Walker (1948). Unrelated and familial cases of mongolism, recorded during recent investigations at the Galton Laboratory, are included. The remaining material was collected by Hanhart (1944) in Switzerland, and the details were very kindly made available to the present writer in 1947 and 1948. Maternal ages of all these familial cases are given in the Appendix: further details on the relationships are available at the Galton Laboratory. The data would have been considerably more extensive if earlier writers had recorded maternal ages in every case. In several instances, information was elicited on this point from the writers themselves by correspondence.

In Table I a pooled sample of 609 male and 429 female cases of mongolism, from widely different sources, is classified according to maternal age. The average for 1038 cases is 36·6 years and the standard deviation is 7·1 years. For comparison, the mean maternal age of all births in England and Wales in 1939 may be quoted, that is 28·6 years with a standard deviation of 6·0 years. In detail the data on mongolism are statistically not quite homogeneous because of an unusually high mean maternal age for females in van der Scheer's material. Nevertheless, the slightly greater mean maternal age of all females (36·97 years) is not significantly higher than that of all males (36·30 years). Brousseau's sample of 584 cases, in which sexes were not differentiated, has a still lower mean of 35·47 years with a standard deviation of 7·99 years.

An interesting point, which deserves comment, is that, in data from many sources, the distribution of maternal ages of mongols tends to be somewhat bimodal. Sometimes there are actually two maxima, one at about 27 and another at about 42 years though, more often, there is merely a rather marked skewness of the distribution.

Taking first of the familial cases the instances of two or more in a sibship, a changed character of the maternal age distribution is obvious (see Appendix). There is a far higher proportion of cases with young mothers than in the control sample, and the mean maternal age of brother and sister pairs, 34·2 years, differs significantly from the mean of the pooled sample. To some extent this reduction in maternal age can be regarded as a natural consequence of having to fit more than one affected case into the sibship. In fact, the phenomena of increased incidence in the sibship and decreased maternal age cannot be clearly separated here.

The distributions of maternal ages in other familial instances are shown in Table II. Here the instances are separated according to whether transmission of the genetical influence can be supposed to have come either through the father or through the mother. FIG. 1 shows the separation diagrammatically. In the case of affected uncle and niece, for example, the sex of the parent of the niece is the relevant factor, but nothing can be inferred about the sex of the transmitting parent of the affected uncle. When cousin pairs are affected, however, information is available concerning the transmitting parents of both cases. The means given in Table III show that the maternal ages of cases with transmission through the paternal line do not differ significantly from those in the control sample. The mean maternal age is 35·8 years for 129 such cases. However, mongols who have affected relatives on the maternal

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side have a maternal age distribution comparable to that in affected sib pairs, with a mean value of 33·0 years for 121 cases. The mean of the pooled sample of the 1,038 cases shown in Table I, to which Brousseau's material has been added, making a total of 1,622 cases, is 36·2 years with a standard deviation of 7·4 years. The difference between these means, 36·2 — 33·0 = 3·2 years, is more than four times its standard error, 0·7 years, and extremely significant

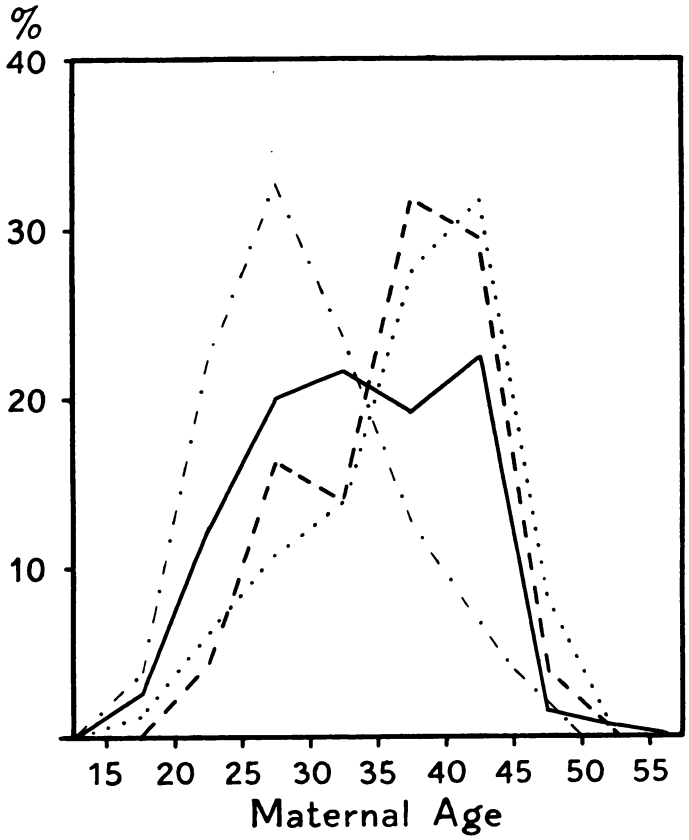


FIG. 1.—Maternal age distributions.
———— Mongolism: transmission through mother (121 instances).
— — — — Mongolism: transmission through father (129 instances).
. . . . . Mongolism: unrelated sample (1,038 cases).
—·—·—·— All births (England and Wales, 1939).

from the statistical point of view. Further inspection of Table III shows that the mean maternal age, 30·0 years for 10 cases, one with an affected mother, five with maternal aunts or uncles affected and four maternal half sibs, is so low as to be indistinguishable from the ordinary value for all births in the population, i.e., 28·6 years in the *Statistical Review for England and Wales* (1939).

Assuming these phenomena to be real and not just chance effects, what light do they throw upon the problem of causation? The lateness of maternal age in cases of mongolism can be perhaps regarded as evidence for a protective

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mechanism whereby a fairly common hereditary trait, which predisposes the embryo to a condition of arrested growth, has been rendered relatively harmless by natural selection. That is to say, most mothers have a quality which preserves their susceptible children from mongolism until a fairly late age is reached. A few mothers might lack this capacity, on account of genetical or other peculiarities, and mongol children could be born to them at any age. If the genetical influence determining mongolism were entirely dependent upon the mother's constitution, this would be manifested by a great excess in collateral relationship between affected cases through the maternal line as compared with those through the paternal line.

If affected cousin pairs are examined separately, a slight excess is found of pairs in which two mothers of mongols are related as compared with those in which two fathers are related, provided that the relationship is fairly close. Examples of paternal line relationships are found in excess in the more distant degrees, possibly because of greater ease in tracing some of them by surnames. On the whole, the data suggest that paternal as well as maternal influences are concerned in the genetics of mongolism. The interesting point, shown in Table IV, that cousin pairs in which the parents of mongols are of like sex (73 pairs) are significantly more frequent than those in which they are of unlike sex (42 pairs), was assumed previously (Penrose, 1947) to be evidence in favour of the assumption that antigenetic incompatibility between mother and foetus might be a causal factor. This theory, however, in the simple form suggested, is disproved by the occurrence of the affected mother and child pair recorded by Lelong *et al.* (1949).

The neatest genetical hypothesis to account for the causation of mongolism would be that a single gene is responsible for the diathesis or susceptible constitution of the foetus. It can then be further supposed that, when the mother also has this diathesis, the foetus has less protection, the chance of producing affected offspring is increased and the maternal age is lowered. The frequency of the foetal diathesis can be roughly estimated by reference to the incidence of mongolism at late maternal ages, that is, 45 years or more ; this is greater than 1 in 40, and may be as high as 1 in 25. Some estimates are even higher and the frequency of 1 in 10 has been suggested by Benda (1947). Though the diathesis would have a frequency of, say, one in 25, its manifestation must be of the right order of magnitude to agree with the overall incidence of mongolism at birth, that is between 1 in 600 and 1 in 700. It is possible, however, that the constitution of mothers of mongols may include the tendency to continue ovulation to a later age than is usual in the general population. If so, the estimates of the frequency of foetal diathesis based on frequency of mongols at very late maternal ages, say 50 years and over, would be misleading. If the causative single gene were supposed to be dominant (Hanhart, 1944), its frequency in the general population would be about half that of the foetal diathesis, say 1 in 50. If it were recessive the gene frequency would be much greater, say 1 in 5. The effects of these two hypotheses are difficult to separate theoretically. On the whole, the low familial incidence in sibships reaching an observed maximal value of less than one quarter even at late maternal ages (Penrose, 1932) points to the recessive

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hypothesis as the more probable. Moreover, though a recessive diathesis in the foetus would be always inherited through both parents, only occasionally would either parent be of the recessive genotype like the foetus. With a gene frequency of 1 in 5, the mother would have the same recessive diathesis as the foetus in one-fifth of the cases. On the additional assumption that the homozygous mothers might give birth to mongols at younger ages than the heterozygous carrier mothers, the bimodal tendency of maternal age distributions of mongols, as shown in Table I, could be accounted for. Agreement with observation is fairly good if the assumption is made that when the mother is homozygous the manifestation of mongolism in the offspring is doubled.

It is unprofitable to enlarge upon all the different hypotheses which might be invented to account for the genetical background of mongolism. The data available at present are sufficient to exclude some theories, but they are inadequate to give confidence to any positive assumption. Although in most data male cases outnumber female cases, there is no hint of sex linkage or partial sex linkage of the hereditary factor as judged by the study of affected relatives. For example, affected sibs, collected in Table V, show no excess of pairs of like sex.

To summarize, then, all available data on familial mongolism in which degree of relationship and maternal age are specified have been assembled. Analysis leads to the suggestion that mongolism could be due to a single very common gene (frequency 1 in 5 in the general population). All homozygous foetuses are susceptible. They have a frequency of 1 in 25, but only about one susceptible case in 27 is actually affected, leading to an absolute incidence of 1 in 675 at birth. Manifestation of the condition is largely controlled by factors connected with maternal age. Maternal age in cases showing inheritance through the mother is significantly lowered. To account for this it is suggested that the defensive effect of young maternal age over the susceptible foetus is reduced when the mother herself is homozygous for the hypothetical gene, that is, when she herself is a mongol or a potential mongol. No suggestions are made about the pathology of the process by which maternal age exerts its peculiar aetiological influence.

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APPENDIX.

LIST OF RELATED CASES OF MONGOLISM GIVING SOURCES, SEXES, MATERNAL AGES AND DEGREES OF RELATIONSHIP DIFFERENTIATING TRANSMISSION THROUGH FATHER AND THROUGH MOTHER.

Previously unpublished instances are identified by reference numbers and/or surname initials: fuller records are available at the Galton Laboratory, University College, London.

TABLE I.—*Distribution of Mongols by Maternal Age.*
(*Control sample of unrelated cases.*)

Source.	Maternal age. 15	20	25	30	35	40	45	50	Total.
Males:									
Galton Laboratory	1	4	12	21	24	36	5	1	104
Penrose	3	11	11	18	52	57	17	0	169
van der Scheer (1927)	1	13	26	22	45	53	20	0	180
Orel (1927)	0	9	8	10	13	17	4	0	61
Hanhart (1948)	0	0	2	8	12	8	0	0	30
Beall and Stanton (1945)	1	5	12	7	19	15	6	0	65
Total males	6	42	71	86	165	186	52	1	609
Females:									
Galton Laboratory	1	1	8	15	28	23	5	0	81
Penrose	0	8	8	13	21	32	9	0	91
van der Scheer (1927)	0	5	11	13	38	54	15	0	136
Orel (1927)	1	3	5	7	14	11	2	0	43
Hanhart (1948)	0	1	2	5	6	8	0	0	22
Beall and Stanton (1945)	2	4	7	7	12	17	7	0	56
Total females	4	22	41	60	119	145	38	0	429
Total males and females	10	64	112	146	284	331	90	1	1,038
Percentage	1·0	6·1	10·8	14·1	27·3	31·9	8·7	0·1	100·0

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TABLE II.—*Distribution of Instances of Mongolism according to Maternal Age-group.*

Central maternal age.	Sample from Table I.		Familial instances: relationship to other affected case.			
			Through father.		Through mother.	
	Number.	Percentage.	Number.	Percentage.	Number.	Percentage.
17	10	1·0	0	0·0	3	2·5
22	64	6·1	6	4·6	15	12·4
27	112	10·8	21	16·3	24	19·8
32	146	14·1	18	14·0	26	21·5
37	284	27·3	41	31·8	23	19·0
42	331	31·9	38	29·4	27	22·3
47	90	8·7	5	3·9	2	1·7
52	1	0·1	0	0·0	1	0·8
Total (all ages)	1,038	100·0	129	100·0	121	100·0

TABLE III.—*Maternal Ages (in years) of Instances of Mongolism with Affected Relatives.*

Relationship of affected relative.		Relationship through father.		Relationship through mother.	
Type.	Degree.	Number of instances.	Mean maternal age.	Number of instances.	Mean maternal age.
Parent, half-sib, uncle, niece, etc.	1/2 to 1/4	7	35·1	10	30·0
First cousin, great uncle, first cousin once removed, etc.	1/8 to 1/16	38	37·0	47	33·2
Second cousin or more distant relative.	1/32 to 1/4,096	84	35·3	64	33·3
(i) All types	1/2 to 1/4,096	129	35·8	121	33·0
(ii) Control sample from Table I.	—	1,038	36·6	1,038	36·6
(iii) Sample from Table I combined with that from Brousseau (1928)	—	1,622	36·2	1,622	36·2
Difference (iii) — (i) with standard error	—	—	0·4±0·7	—	3·2±0·7

TABLE IV.—*Cousin Pairs Classified by Degree of Relationship and Sex of Transmitting Parent.*

Degree of relationship of mongols.*	Sex of transmitting parent.		
	Both male.	One male, one female.	Both female.
1/8	7	15	10
1/16	3	1	5
1/32	3	3	5
1/64	6	5	3
1/128	4	5	3
1/256	4	6	3
1/512	6	4	3
1/1,024	2	2	2
1/2,048	3	0	0
1/4,096	1	1	0
Totals	39	42	34

* First cousins correspond to 1/8, first cousins once removed to 1/16, second cousins to 1/32, etc.

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TABLE V.—*Affected Sib Pairs Classified by Sex.*

Brother and brother.	Brother and sister.	Sister and sister.	Total.
11	23	8	42

Affected Brothers and Sisters.

Source.	Sex and maternal age.				Degree of relationship.	Notes.
Glassburg (1924)	m 34		m 37		1/2	—
Fantham (1925)	f 23	f 31	m 34		1/2	3 pairs.*
Borovsky (1928)	f 25		f 28		1/2	—
Johnson (1936)	m 27		f 43		1/2	—
,, (1936)	m 26		f 30		1/2	—
MacKaye (1936)	m 40		m 40		1/2	Dizygotic twins.
Péhu and Gaté (1937)	m 23	m 25	f 27	f 29	1/2	6 pairs.
Tatafiore (1937)	f 27		f 34		1/2	—
Geyer (1939)	m 19		m 20		1/2	—
Auden (1947)	m 21		f 22		1/2	—
Benda (1947)	m 38		m 41		1/2	—
,, (1947)	m 26	f 31	m 35		1/2	3 pairs.
Keith (1948)	f 33		m 39		1/2	*
Engler (1949)	m 40		f 43		1/2	*
Jervis (1943)	m 39		m 39		1/2	Dizygotic twins.
Halperin (1939)	f 39		m 40		1/2	—
Ford-Walker (1948)	m 40		m 43		1/2	—
Penrose 1, 2	m 38		f 42		1/2	—
,, 3, 4	m 33		f 38		1/2	—
,, 5, 6	m 37		m 42		1/2	
,, 13, 14	m 40		m 45		1/2	—
,, 23, 24	m 20		f 23		1/2	—
,, 25, 26	f 38		m 42		1/2	—
,, 27, 28	f 23		f 32		1/2	—
,, 41, 42	m 31		f 44		1/2	—
Hanhart 1 TMSHR	f 31		m 38		1/2	—
,, 59 GD	f 30		m 40		1/2	—
,, 60 BH	m 41		m 42		1/2	—
,, 61 WM	f 39		f 42		1/2	—
,, 62 M	f 39		f 43		1/2	—
Galton Laboratory 18, 19 S	m 43		f 49		1/2	—
,, ,, 154, 155 K	f 26		f 32		1/2	—
,, ,, 212 G	m 32		f 37		1/2	—

Asterisk (*) indicates that maternal ages were ascertained by subsequent investigation.

Cases with Affected Parent, Uncle or Aunt, Great Uncle or Great Aunt, or Half Sib.

Source.	Sex and maternal age.				Degree of relationship.	Notes.
Fantham (1925)	f 23	f 31	m 34		1/4	Father's sib affected.
Hanhart 17 FRW		f 45			1/4	,, ,, ,,
,, 18 WB		f 38			1/4	,, ,, ,,
,, 19 REK		f 35			1/4	,, ,, ,,
Anonymous (1950)		m 40			1/4	,, ,, ,,
Penrose 37, 38		m 32			1/8	,, half-sib affected.
,, 39, 40		m 42			1/8	,, aunt affected.
Lelong *et al.* (1949)		f 30			1/2	Mother affected.
Doxiades and Portius (1938)		m 19			1/4	Mother's sib affected.
Hanhart 58 F		f 34			1/4	,, ,, ,,
Galton Lab. 185, 187 TB		f 37			1/4	,, ,, ,,
Anonymous (1950)		f 39			1/4	,, ,, ,,
Penrose 11, 12		m 22			1/4	,, ,, ,,
,, 33, 34		m 42			1/8	,, uncle affected.
Lahdensuu (1937)	f 25		m 35		1/4	Maternal half-sibs.
Hanhart 57 MM	m 29		m 33		1/4	,, ,, ,,

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Pairs of Affected Cousins.

(A) Father of (i) related to Father of (ii).

Source.	Sex and maternal age. (i)	(ii)	Degree of relationship.
Lipnitzky and Boshes (1942)	f 45	m 34	1/8
Ford-Walker (1948)	f 28	m 36	1/16
,,	f 28	f 34	1/16
,,	f 34	m 36	1/8
Penrose 9, 10	f 45	m 34	1/32
,, 21, 22	m 38	m 44	1/8
,, 31, 32	f 41	m 34	1/8
Hanhart 1 TMSRH	f 42	f 31	1/64
,,	f 42	m 38	1/64
,, 2 HLS	m 41	m 23	1/2,048
,,	m 41	m 43	1/1,024
,,	m 23	m 43	1/64
,, 3 WES	m 24	f 29	1/512
,,	m 24	m 43	1/512
,,	f 29	m 43	1/512
,, 4 GHH	f 39	m 40	1/512
,,	f 39	f 41	1/512
,,	f 14	m 40	1/8
,, 5 RJ	f 28	f 32	1/256
,, 6 AZ	m 40	m 41	1/256
,, 7 LB	m 36	m 37	1/256
,, 8 HH	f 42	f 33	1/64
,, 9 TE	f 31	m 41	1/64
,, 10 S	m 41	m 35	1/8
,, 11 G	m 42	m 42	1/32
,, 12 F	f 40	m 37	1/8
,, 13 SWW	f 37	f 49	1/32
,, 14 DW	f 38	m 35	1/16
,, 15 SG	f 27	f 41	1/128
,, 16 OK	f 36	m 25	1/128
,, 42SSME	m 43	f 32	1/128
,, 43 MA	f 28	m 26	1/128
,, 45 F	m 39	m 36	1/256
,,	m 39	m 36	1/512
,,	m 39	m 25	1/2,048
,,	m 36	m 37	1/64
,,	m 36	m 25	1/1,024
,,	m 25	m 37	1/2,048
,, 46 HTW	f 30	f 38	1/4,096

(B) Mother of (i) related to Mother of (ii).

Source.	Sex and maternal age. (i)	(ii)	Degree of relationship.
Chotzen (1925)	f 48	f 30	1/8
Borovsky (1928)	f 25	f 39	1/8
,,	f 28	f 39	1/8
Penrose (1938)	m 21	f 27	1/8
,,	m 21	f 24	1/8
,,	m 21	f 21	1/16
,,	f 27	m 24	1/8
,,	f 27	f 21	1/16
,,	m 24	f 21	1/16
Penrose 7, 8	m 50	m 38	1/8
,, 37, 36	m 39	m 41	1/8
Galton Laboratory 109, 111 G	m 40	m 30	1/32
,, ,,	f 43	m 40	1/32
,, ,,	f 43	m 30	1/32
,, ,, 102, 94 T	m 45	m 44	1/16
,, ,, 213 S	m 40	f 34	1/32
Hanhart 1 TMSRH	f 31	m 33	1/64
,,	m 38	m 33	1/64

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Pairs of Affected Cousins—cont.

(A) Father of (i) related to Father of (ii).

Source.	Sex and maternal age. (i)	(ii)	Degree of relationship.
,, 44 MGS	f 40	m 35	1/8
,, 47 KRP	f 30	f 42	1/1,024
,,	f 30	f 19	1/1,024
,,	f 42	f 19	1/128
,, 48 GKH	m 27	f 43	1/128
,,	m 27	m 24	1/512
,,	f 43	m 24	1/512
,, 49 KSSS	m 43	m 31	1/16
,, 50 K	m 32	f 30	1/8
,, 51 B-U	m 27	f 39	1/256
,, 52 KB	f 38	m 25	1/256
,, 53 LB	m 34	m 21	1/256
,, 54 MAE	m 24	f 42	1/64
,, 55 ZW	m 39	f 42	1/64
,, 56 SW	f 32	m 35	1/128
,, 44 MGS	f 33	f 26	1/512

(C) Father of (i) related to Mother of (ii).

Source.	Sex and maternal age. (i)	(ii)	Degree of relationship.
Anonymous (1950)	m 40	f 39	1/8
Galton Laboratory 109 G	f 43	f 40	1/8
Penrose 19, 21	m 39	f 43	1/8
,, 29, 30	f 23	m 44	1/32
Hanhart 1 TMSRH	f 31	f 36	1/128
,,	m 38	f 36	1/128
,, 20 HA	m 28	m 26	1/32
,, 21 DP	m 40	f 37	1/8
,, 22 RH	f 42	f 28	1/8
,, 23 HA	m 41	f 38	1/256
,, 24 GT	m 29	m 34	1/128
,, 25 OB	f 45	f 42	1/256
,, 26 KK	m 41	m 30	1/64
,, 27 RR	m 45	m 39	1/64
,, 28 SR	f 38	f 28	1/64
,, 29 NS	m 29	f 34	1/8
,, 30 VC	f 39	f 35	1/8
,, 31 BH	m 43	f 34	1/8
,, 32 G	m 37	m 36	1/8
,, 33 FFR	m 41	m 35	1/8
,, 34 KR	f 43	f 31	1/8
,, 35 SD	f 31	f 33	1/16
,, 36 MPSS	f 40	m 36	1/64
,, 37 LHND	m 41	f 40	1/128
,, 38 WR	m 35	f 42	1/8
,, 39 MHS	m 42	m 20	1/8
,, 39 MHS	m 42	m 20	1/8
,, 40 GHRV	f 35	m 42	1/64
,, 41 GZ	m 27	f 29	1/512
,, 42 SJME	f 32	f 27	1/512
,, 43 MA	f 28	f 33	1/256
,, 44 MGS	f 36	f 40	1/8
,,	f 36	m 35	1/8
,, 45 F	m 39	m 27	1/1,024
,,	m 36	m 27	1/512
,,	m 25	m 27	1/1,024
,,	m 37	m 27	1/256
,, 46 HTW	f 30	m 44	1/32
,,	f 38	m 44	1/4,096
,, 42 SJME	m 43	f 27	1/512
,, 43 MA	f 28	f 26	1/256
,,	m 26	f 33	1/256
,,	m 26	f 26	1/128

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