Supplementary Table 2: spontaneous mouse mutations resulting in experimentally relevant immune phenotypes.

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
Btk	Bruton agammaglobulinemia tyrosine kinase	X	tyrosine-protein kinase; B cell development, differentiation, and activation; BCR signal transduction; regulation TLRs signaling in B cells; high-affinity IgE receptor signal transduction on mast cell; regulation of neutrophilic granulocyte maturation and function.	*Btk*^{xid}	X-linked immune deficiency	single point mutation; C-to-T transition; arginine to cysteine substitution at position 28; LOF; X-linked recessive inheritance.	CBA/HN	Immune: homozygous females and hemizygous males; defective B cell development, differentiation, and activation (including T-independent B lymphocytes); profound B-1 cell deficiency; impaired IgM and IgG production in response to thymus-independent TI-2 antigens (impaired T-independent B cell response); impaired granulocyte maturation and function; abnormal regulation of TLRs signaling; defective mast cells degranulation and proinflammatory cytokines production upon high-affinity IgE receptor engagement; severe hypogammaglobulinemia (especially IgM and IgG3).[1-5] Other: N/A.	Bruton-type agammaglobulinemia; X-linked immunodeficiency
Cacnb4	calcium channel, voltage-dependent, beta 4 subunit	2	cytoplasmic β subunit of voltage-dependent calcium channels; important role in calcium channel function by modulating G protein inhibition, increasing peak calcium current, controlling the α_1 membrane pore subunit, and shifting the voltage dependence of activation/inactivation.	*Cacnb4*^{lh}	lethargic	insertion mutation; 4 bp; splice donor site; exon skipping; translational frameshift; protein truncation with loss of the α_1 subunit binding site; LOF; hypomorphic; recessive inheritance.	BALB/cGn	Immune: immunodeficiency possibly resulting from a stress response; early thymic involution at 3 to 4 weeks; reduced lymphoid organs size; severe leukocytopenia and anemia; defective cell-mediated immune functions; increased levels of serum IgG1.[6-9] Other: early lethality (approx. 50 days of age); ataxia and lethargy followed by seizures; absence epilepsy (petit mal seizures); no evidence of pathological changes in the CNS or skeletal muscles; dyskinetic motor behavior; pituitary-adrenal hypercorticism.[9-11]	idiopathic generalized epilepsy (IGE); juvenile myoclonic epilepsy (JME); episodic ataxia, type 5
Csf1	colony stimulating factor 1	3	cytokine; differentiation/maturation and activation of monocytes and Mφs; maintenance of bone marrow progenitor cells; innate immunity; inflammation.	*Csf1*^{op}	osteopetrosis	insertion mutation; single nucleotide (T); 262 bp downstream from the initiation codon; frameshift with premature stop codon; LOF; recessive inheritance.	B6;DW-*Pou1f1*^{dw}	Immune: perturbed bone marrow microenvironment with defective hematopoiesis; impaired differentiation and survival of cells of the mononuclear phagocyte system; impaired B lymphopoiesis; generalized deficiency in normal tissue-resident Mφs including osteoclasts, microglia and Kuppfer's cells; attenuated tumorigenesis due to defects in development of pro-tumorigenic M2 polarized TAMs.[9,12-15] Other: high rate of lethality around weaning; severe growth retardation; osteopetrosis resulting in abnormal skeletal development; craniofacial deformities (shortened snout and domed skull); unerupted incisors; shortened extremities; optic nerve compression; hearing impairment due to abnormal ossicle formation and reduced tympanic bullae volume; obliteration of bone marrow cavity; dysfunctional satellite cells in skeletal muscles; reduced fertility in females; defective mammary gland development and lactation; no phenotype rescue following transplant of normal bone marrow cells (not intrinsic defect of hematopoietic cells).[9,16-18]	osteopetrosis
Dock2	dedicator of cytokinesis 2	11	guanine nucleotide exchange factor; regulation of Rac signaling; chemotaxis; leukocyte response to chemokines; cytoskeletal rearrangements; lymphocyte migration; plasmacytoid DC homing and function.	*Dock2*^{m1Hsd}	mutation 1, Harlan	duplication and nucleotides substitution; duplication of exons 28 and 29; frameshift mutation after exon 29 with premature stop codon; LOF; hypomorphic; recessive inheritance.	C57BL/6NHsd (from specific breeding facilities)	Immune: (compared to other C57BL/6 substrains including C57BL/6NClr, C57BL/6NJ or C57BL/6NTac) increased numbers of CD8+ T cells; decreased follicular B cell number; impaired development of splenic marginal zone B cells; abnormal activation of B cells; defective development and function of plasmacytoid DC.[19,20] Other: hearing impairment; cochlear degeneration.[21]	immunodeficiency 40
Eef1a2	eukaryotic translation elongation factor 1 alpha 2	2	α subunit of the elongation factor-1 complex; tissue specific (e.g. neurons, cardiac and skeletal muscle); enzymatic delivery of aminoacyl tRNAs to the ribosome.	*Eef1a2*^{wst}	wasted	intragenic deletion; promoter and first exon of the gene; LOF; recessive inheritance.	HRS/J	Immune: immunodeficiency likely resulting from a stress response; marked lymphoid atrophy/depletion in the spleen, thymus, and lymph nodes; leukopenia; defects in mucosal immunity resulting from reduced number of IgA plasma cells in the entire large and small intestine; decreased number of DP thymocytes; increased sensitivity of T cells to ionizing	ataxia telangiectasia

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
								radiation; cytokine imbalance (decreased IL5 and increased IL1, IL2, IFNγ, and TGFβ1);[9,22,23] Other: early lethality (approx. 30 days of age); tremors and ataxia at weaning; progressive motor neuron degeneration and loss accompanied by gliosis, generalized muscle wasting and paralysis;[9,23,24]	
Fas	TNF receptor superfamily member 6	19	death receptor ; extrinsic apoptotic pathway; peripheral tolerance; AICD; cytotoxic T cell and NK cell-mediated apoptosis.	*Fas*^{lpr} *Fas*$^{lpr\text{-}cg}$	*Fas*^{lpr}*:* lymphoproliferation *Fas*$^{lpr\text{-}cg}$*:* lymphoproliferation complementing gld	*Fas*^{lpr}*:* transposon insertion; ETn; intron 2; premature termination of transcription; aberrant mRNA splicing; leaky mutation; low levels of full-length transcripts and functional receptor formation; LOF; hypomorphic; recessive inheritance. *Fas*$^{lpr\text{-}cg}$*:* single point mutation; T-to-A transversion; replacement of a highly conserved isoleucine with an asparagine; extracellular domain; LOF; recessive inheritance.	*Fas*^{lpr}*:* MRL/MpJ *Fas*$^{lpr\text{-}cg}$*:* CBA/KlJms	Immune: generalized autoimmune and lymphoproliferative disorder; thymus (T cell) dependent (neonatal thymectomy associated with phenotype rescue); progressive splenomegaly and lymphadenopathy; secondary lymphoid organs with expansion of DN and B220+ T cells; defective CTL activity; autoimmunity; loss of AICD; unregulated B cell activation in GC; multisystemic infiltrate of activated lymphoid cells; production of autoantibodies including anti-DNA and anti-IgG; immune complex GN; polyarthritis; vasculitis; dysfunctional M1 Mφ-mediated tumor immunity; *Fas*$^{lpr\text{-}cg}$ autoimmune phenotype less severe than *Fas*^{lpr} mice.[25-31] Other: defective neuronal branching; impaired regulation of spermatogenesis.[32-34]	autoimmune lymphoproliferative syndrome; Sjogren's syndrome; systemic lupus erythematosus
Fasl	Fas ligand	1	death receptor ligand; extrinsic apoptotic pathway; peripheral tolerance; activation-induced cell death; cytotoxic T cell and NK cell-mediated apoptosis.	*Fasl*^{gld}	generalized lymphoproliferative disease	single point mutation; T-to-C transition; replacement of a highly conserved phenylalanine with a leucine; LOF; recessive inheritance.	C3H/HeJ	Immune: generalized autoimmune and lymphoproliferative disorder; thymus (T cell) dependent (neonatal thymectomy associated with phenotype rescue); progressive splenomegaly and lymphadenopathy; secondary lymphoid organs with expansion of DN and B220+ T cells; defective CTL activity; autoimmunity; loss of AICD; unregulated B cell activation in GC; multisystemic infiltrate of activated lymphoid cells; production of autoantibodies including anti-DNA and anti-IgG; immune complex GN; polyarthritis; vasculitis.[25-27,35,36] Other: impaired neuronal branching; impaired regulation of spermatogenesis; enhanced intestinal tumorigenesis with reduced neutrophilic influx.[32,34,37]	autoimmune lymphoproliferative syndrome; systemic lupus erythematosus
Fcer2a	Fc receptor, IgE, low affinity II, alpha polypeptide	8	CD23, low-affinity receptor for IgE, negative regulation of IgE levels, allergy and resistance to parasites.	*Fcer2a*^{Hie}	hyper IgE	multiple point mutations; decreased levels of membranous expression; LOF; hypomorphic; dominant inheritance.	NZB, 129P1/ReJ, 129/SvJ	Immune: increased levels of IgE; exaggerated IgE response; enhanced resistance and protection against parasitic infestations.[38,39] Other: N/A.	N/A
Foxn1	forkhead box N1	11	Forkhead/winged-helix family of transcription factors; differentiation of keratinocytes; development of the thymus; regulation of keratin gene expression; regulation of genes involved in antigen processing and thymocyte selection.	*Foxn1*^{nu}	nude	intragenic deletion; single bp; exon 3; frameshift with premature stop codon; LOF; recessive inheritance.	albino stock	Immune: thymus agenesis/dysgenesis; developmental failure of the thymic anlage; cystic changes of the thymic epithelial rudiments; reduced numbers of mature and functional T cells; extrathymic T cell lymphopoiesis with functional T cells development (especially in old mice exposed to “dirty” environment); partial defect in B cell development and function due to absence/paucity of functional T helper cells; increased number and cytotoxic capability of NK cells and Mφs; normal intraepithelial γδ T cell repertoire at mucosal sites and skin;[9,40-44] autoimmunity; production of autoantibodies including anti-nuclear antibodies; systemic immune complex disease with glomerular involvement;[9,45] high susceptibility to infection by a broad spectrum of bacterial and viral pathogens;[42,46]	DiGeorge syndrome; T cell immunodeficiency; congenital alopecia

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
								Other: grossly hairless (hence the term "nude"); normally cycling hair follicles; defects in follicular and epidermal keratinization; impaired hair shaft formation and eruption;[9,47,48] poor breeders; males with decreased serum levels of LH and FSH and impaired spermatogenesis; females with decreased serum levels of estradiol, progesterone, and thyroxine.[9,49,50]	
Foxp3	forkhead box P3	X	forkhead/winged-helix family of transcriptional regulators; development and function of regulatory T cells; immune tolerance (especially self-tolerance).	*Foxp3^{sf}*	scurfy	insertion mutation; 2 adenosine residues into exon 8, frameshift with premature stop codon; truncated protein lacking the carboxy-terminal forkhead domain; LOF; X-linked recessive inheritance.	MR stock	Immune: scaling and crusting dermatitis; CD4+ T cell-mediated autoimmune disorder; infiltrations of activated immune cells consisting of lymphocytes, DCs, Mφs, and eosinophils into several organs including skin, lungs, kidneys and liver; marked enlargement of peripheral lymph nodes and splenomegaly with proliferation/infiltration of activated lymphocytes, DCs, and Mφs; autoimmune hemolytic anemia; hypergammaglobulinemia; inability to properly regulate CD4+ T cell activation and function; overexpression of proinflammatory cytokines; impaired peripheral immune tolerance.[51-53] Other: failure to thrive; early lethality (approx. 20-50 days of age); male infertility; significantly reduced expression of pituitary gonadotropins; arrested spermatogenesis; lack of transcriptional regulation of ErbB2 oncogene with increased incidence of spontaneous mammary carcinomas; impaired megakaryopoiesis.[54-56]	immune dysregulation-polyendocrinopathy-enteropathy-X-linked syndrome (IPEX); Wiskott-Aldrich syndrome
Hc	hemolytic complement	2	complement system; innate immunity; C5a anaphylatoxin with spasmogenic and chemotactic activity; C5b subunit of the membrane attack complex.	*Hc^{0}*	hemolytic complement deficient	intragenic deletion; 2 bp deletion near the 5' end of the gene; frameshift with premature stop codon 4 bases after the deletion; truncated transcript; LOF; recessive inheritance.	A/HeJ, AKR/J, DBA/2J, NZB/B1NJ, SWR/J, and B10.D2/oSnJ	Immune: impaired leukocyte chemotaxis; impaired bacterial and fungal clearance during opportunistic infections; defective granulomatous response; increased susceptibility to viral infections and DSS-induced colitis; decreased susceptibility to CIA, experimental airway inflammation, EAE, and autoimmune GN.[57-68] Other: impaired liver regeneration; decreased susceptibility to reperfusion injury; delayed bile duct ligation-induced hepatobiliary fibrosis; delayed fracture healing.[69,70]	complement component 5 deficiency (C5D)
Hr	hairless	14	histone demethylase; demethylation of Lys9 on histone H3; transcriptional corepressor of multiple nuclear receptors including thyroid hormone receptor, the retinoic acid receptor-related orphan receptors, and the vitamin D receptors; regulation of hair cycle, epidermal maturation, neural activity, and cell cycle.	*Hr^{hr}* *Hr^{rh}*	*Hr^{hr}*: hairless *Hr^{rh}*: rhino	*Hr^{hr}*: retroviral integration; intron 6; leukemia proviral sequences; aberrant splicing; LOF; recessive inheritance. *Hr^{rh}*: unknown mutation; mutant Hr^{rh} allele identified by a negative complementation test with hr^{hr} phenotype.	Hr^{hr}: HRS/J Hr^{rh}: RHJ/LeJ	Immune: early thymic cortical atrophy; impaired splenic T cell activation and expansion; defective splenic response to T-dependent antigens; defective Mφ activation; autoimmunity; hypergammaglobulinemia; production of autoantibodies including anti-nuclear antibodies; systemic immune complex disease with glomerular, hepatic, splenic, and skin involvement.[9,71-73] Other: normal first hair cycle followed by rapid and complete hair loss/alopecia including vibrissae and cilia (eyelashes); dysregulation of catagen phase of hair cycle; failure of trichilemmal keratinization in the developing club hair; abnormal pylosebaceous units; hair follicle degeneration into utriculi and deep dermal cysts; sebaceous gland hyperplasia associated with deep dermal cysts; pyogranulomatous dermatitis; severe epidermal thickening/hyperplasia (mainly associated with the Hr^{rh} mutation); high incidence of thymic lymphoma; increased sensitivity to UV and chemically induced skin tumors.[9,71,74]	alopecia universalis congenita; atrichia with papular lesions; Marie Unna hereditary hypotrichosis
Il2	interleukin 2	3	secreted cytokine; clonal expansion of B and T cells; immune cell activation including NK cells; development of regulatory T cells, effector T cells, and	*Il2^{m1}*	mutation 1	undefined mutation; hypoactive variant *Il2* gene; smaller polyglutamine tract with shortening of the binding site; LOF;	MRL/MpJ, SJL/J, and NOD/ShiLtJ	Immune: defective T cell activation; impaired AICD with decreased susceptibility to Fas-mediated apoptosis; predisposition to autoimmune conditions.[75] Other: N/A.	N/A

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
			memory T cells; immune tolerance; adaptive cell-mediated immunity.			hypomorphic; recessive inheritance.			
Kit	KIT proto-oncogene receptor tyrosine kinase	5	receptor tyrosine kinase protein; regulation of haematopoietic stem cell survival, differentiation, and proliferation; gametogenesis; mast cell development, migration, and function; melanogenesis.	*Kit^{W}* *$Kit^{W\text{-}v}$*	*Kit^{W}:* dominant spotting *$Kit^{W\text{-}v}$:* viable dominant spotting	*Kit^{W}:* single point mutation; G-to-A transition; disrupted splice donor site; exon skipping; aberrantly spliced transcripts; LOF; semidominant inheritance. *$Kit^{W\text{-}v}$:* C-to-T transition at nucleotide 2007; threonine to methionine substitution at codon 660; LOF; semidominant inheritance.	*Kit^{W}:* C57BL/6J, WB/ReJ *$Kit^{W\text{-}v}$:* C57BL/6J, MWT/Le, NFR/N, NFS/N, and WB/ReN *$Kit^{W}/Kit^{W\text{-}v}$* (compound mutant): WB/ReJ × C57BL/6J F_1 ($WBB6F_1$) hybrid background	Immune: hematologic disorders, including macrocytic anemia, due to impaired pluripotent hematopoietic stem cell differentiation and expansion; impaired resistance to parasitic infection; mast cells deficiency.[9,76-78] Other: early lethality (approx. 7 days of age) in Kit^{W} homozygotes; $Kit^{W\text{-}v}$ homozygotes survive till maturity; defective gametogenesis due to impaired maturation of primordial germ cells during embryonic development; defective melanogenesis due to impaired melanoblasts migration and differentiation during embryonic development; homozygous mutants with diffusely unpigmented coat and normal eye pigmentation; heterozygous mutant with white spotting of the coat; hearing impairment due to abnormal development of intermediate cells in the stria vascularis.[9,76,79-82]	macrocytic anemia
Lep	leptin	6	hormone; endocrine regulation of energy balance; central appetite-regulating factor; neutrophil activation and chemotaxis; Mφ phagocytosis; NK cytotoxicity; TH1 immune response; lymphocyte development.	*Lep^{ob}*	obese	single point mutation; C-to-T transition; codon 105; premature termination of the transcript; LOF; recessive inheritance.	C57BL/6J, C57BL/KsJ	Immune: decreased neutrophil activation and chemotaxis; impaired Mφ phagocytic capacity; defective DC development and maturation; deficient TH1 immune response; defective B and T cells development and survival; impaired NK cell cytotoxicity; reduced inflammatory mediators synthesis; enhanced starvation-induced immunosuppression; reduced susceptibility to experimental autoimmune conditions.[83-88] Other: hyperphagy; obesity; type II diabetes mellitus; osteosclerosis/increased bone mass; hypogonadism.[89,90]	obesity, type 2 diabetes mellitus, metabolic syndrome
Lepr	Leptin receptor	4	hormone receptor; endocrine regulation of energy balance; central appetite-regulating factor; neutrophil activation and chemotaxis; Mφ phagocytosis; NK cytotoxicity; TH1 immune response; lymphocyte development.	*$Lepr^{db}$*	diabetes	single point mutation, G-to-T transversion; abnormal splicing with premature termination of the transcript; loss of signal transduction function; LOF; recessive inheritance.	C57BL/KsJ	Immune: decreased neutrophil activation and chemotaxis; impaired Mφ phagocytic capacity; defective DC development and maturation; deficient TH1 immune response; defective B and T cells development and survival; impaired NK cell cytotoxicity; reduced inflammatory mediators synthesis; enhanced starvation-induced immunosuppression; reduced susceptibility to experimental autoimmune conditions.[85,86,91-93] Other: hyperphagy; obesity; type II diabetes mellitus; osteosclerosis/increased bone mass; hypogonadism.[90,94,95]	obesity, type 2 diabetes mellitus, metabolic syndrome
Lyst	lysosomal trafficking regulator	13	regulation of lysosome/endosomes intracellular trafficking.	*$Lyst^{bg\text{-}J}$*	beige Jackson	intragenic deletion; 3 bp, exon 54, codon 3741; highly conserved isoleucine loss, carboxy terminus; LOF; recessive inheritance.	C57BL/6J	Immune: immune cell and leukocyte dysfunction; disorder of lysosome/secretory granule formation and function in different cell types including granulocytes, Mφs, NK, and cytotoxic T cells; evidence of giant lysosomes/secretory granules in granulocytes, lymphocytes, and mast cells; defective leukocyte chemotaxis; impaired phagolysosome formation; impaired MHC class II antigen presentation; defective leukocyte lytic granules exocytosis and release of bactericidal enzymes; increased susceptibility to opportunistic bacterial infections.[96-99] Other: oculocutaneous hypopigmentation; abnormal enlargement, clumping, and distribution of melanin granules; evidence of giant lysosomes/secretory granules in various tissues/cell types including hepatocytes, renal proximal tubules, neurons, exocrine and endocrine pancreas, thyroid follicles, and type II pneumocytes; anemia and hemorrhagic diathesis due to dysfunction of platelet's α and δ granules; progressive neurological disorder accompanied by nearly complete loss of Purkinje cells.[99]	Chediak-Higashi syndrome

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
Map3k14	mitogen-activated protein kinase kinase kinase 14	11	serine/threonine protein-kinase; promotion of canonical and non-canonical NF-κB signaling cascade; development of secondary lymphoid organs.	*Map3k14^{aly}*	alymphoplasia	single point mutation; G-to-A transition; replacement of a highly conserved glycine for an arginine at position 855; LOF; recessive inheritance.	C57BL/6J	Immune: generalized aplasia of secondary lymphoid organs including lymph nodes, splenic white pulp, Peyer's patches, and isolated lymphoid follicles; lack of normal corticomedullary differentiation in the thymus; high susceptibility to opportunistic infections; defects of somatic hypermutation and immunoglobulin class switching; hypogammaglobulinemia; impaired humoral and cell-mediated immune response; progressive autoimmune condition with CD4+ T cell-rich infiltrates affecting exocrine glands (e.g. pancreas, salivary glands, and lacrimal glands) and other organs.[100-107] Other: impaired mammary gland development and lactation.[108]	Sjogren's syndrome
Mitf	melanogenesis associated transcription factor	6	transcription factor; master regulator of eye and neural crest development; fate and differentiation of melanocyte, RPE, osteoclast, and mast cell; regulation of tyrosinase and tyrosinase-related protein 1 in melanocytes.	*Mitf^{Mi}*	microphthalmia	intragenic deletion; highly conserved arginine loss; impaired DNA binding; LOF; semidominant inheritance.	unspecified	Immune: impaired monocyte/Mφ, NK cell, basophil, and mast cell function; decreased Mφ chemotaxis.[9,109,110] Other: alteration of skin and eye pigmentation; white spotting on the head, tail, and belly, reduced iris pigment (heterozygous mutants); complete absence of skin and eye pigmentation, lack of incisors (homozygous mutants); inner ear defects and deafness; colobomatous micropthalmia; impaired development of dorsal root ganglia and adrenal medulla;[9,110] osteopetrosis; dysfunctional osteoclasts due to cathepsin K and TRAP downregulation (both transcriptional target of Mitf); impaired endocrine control of calcium and phosphate metabolism/homeostasis.[9,111-113]	Waardenburg syndrome type 2A; Tietz albinism-deafness syndrome; coloboma; osteopetrosis; microphthalmia; macrocephaly; albinism; deafness
Mitf	melanogenesis associated transcription factor	6	transcription factor; master regulator of eye and neural crest development; fate and differentiation of melanocyte, RPE, osteoclast, and mast cell; regulation of tyrosinase and tyrosinase-related protein 1 in melanocytes.	*Mitf$^{mi\text{-}vit}$*	vitiligo	single point mutation; G-to-A transition; codon 222; aspartate to asparagine substitution in the helix 1 region of the protein; LOF; recessive inheritance.	C57BL/6J	Immune: decreased number of Langerhans' cells in the interfollicular epidermis; attenuation of allergic contact dermatitis; decreased number of peritoneal mast cells. Other: congenital white spotting of the back and abdomen; progressive depigmentation with each hair molt resulting in complete pigment loss by 2 years of age; degeneration of follicular melanocytes without inflammation; iris pigment dispersion; degeneration and loss of melanocytes within the choroid; expansion of dysfunctional RPE cells with progressive retinal degeneration.[9,114-116]	vitiligo
Mx1	MX dynamin-like GTPase 1	16	Mx GTPase protein family; innate immunity; IFNα an IFNβ-mediated response to intranuclear viruses including orthomyxoviruses and Thogoto virus; inhibition of viral replication.	*Mx1^{s1}* *Mx1^{s2}* *Mx1^{r}*	*Mx1^{s1}*: myxovirus susceptibility 1 *Mx1^{s2}*: myxovirus susceptibility 2 *Mx1^{r}* : myxovirus resistant	*Mx1^{s1}*: exon 9 to 11 deletion compared to the resistant (*Mx^{r}*) allele; LOF; recessive inheritance. *Mx1^{s2}*: exon 10; nonsense mutation resulting in a null allele; LOF; recessive inheritance.	*Mx1^{s1}*: most inbred strains including A/J, BALB/cJ, C57BL/6J, C3H/HeJ, and DBA/2J. *Mx1^{s2}*: CBA/J, CE/J, I/LnJ, and PERA/Ei. *Mx^{r}:* A2G, SL/NiA, T9, and CAST/Ei.	Immune: (compared to feral mice carrying the *Mx1^{r}* allele) increased susceptibility to viral infections including orthomyxoviruses; ineffective IFNα an IFNβ-mediated response to viral infections.[117,118] Other: N/A.	susceptibility to viral diseases including orthomyxoviral infectons
Mx2	MX dynamin-like GTPase 2	16	Mx GTPase protein family; innate immunity; IFNα an IFNβ-mediated response to intracytoplasmic viruses including vesicular stomatitis virus and bunyaviruses (e.g. La Crosse virus and Hantaan virus); inhibition of viral replication.	unspecified	unspecified	insertion mutation; open reading frames (ORFs); single nucleotide (C) at position 1366 of Mx2 mRNA; frameshift with premature termination of the transcript; LOF; recessive inheritance.	functional only in some feral mice (i.e. *Mus musculus musculus* and *Mus spretus*)	Immune: (compared to feral mice expressing the functional form of *Mx2*) increased susceptibility to viral infections including vesicular stomatitis virus and hantaviruses; ineffective IFNα an IFNβ-mediated response to viral infections.[118-120] Other: N/A.	susceptibility to viral diseases including vesicular stomatitis and hantaviruses infections

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
Oas1b	2'-5' oligoadenylate synthetase 1B	5	oligoadenylate synthetase family; dsRNA-activated antiviral enzyme; innate immunity; IFNα and IFNβ-mediated response to viral infections; synthesis of 2',5'-oligoadenylates (2-5As); activation of latent ribonuclease L (RNase L); viral and endogenous RNA degradation; inhibition of viral replication.	*Oas1b*$^{Flv\text{-}mr}$ *Oas1b*$^{Flv\text{-}s}$ *Oas1b*$^{Flv\text{-}r}$	*Oas1b*$^{Flv\text{-}mr}$: minor flavivirus resistance *Oas1b*$^{Flv\text{-}s}$: flavivirus susceptibility *Oas1b*$^{Flv\text{-}r}$: flavivirus resistance	*Oas1b*$^{Flv\text{-}mr}$: nucleotide substitutions; 14 amino acid substitutions; LOF; hypomorphic; dominant inheritance. *Oas1b*$^{Flv\text{-}s}$: single point mutation; C-to-T transition; premature stop codon; protein lacking 30% of the C-terminal region; LOF; dominant inheritance.	*Oas1b*$^{Flv\text{-}mr}$: wild-derived strain MOLD/RkJ *Oas1b*$^{Flv\text{-}s}$: most of the laboratory mouse strains *Oas1b*$^{Flv\text{-}r}$: most wild-derived strains including BSVR, BRVR, CASA/RK, and CAST/Ei.	Immune: (compared to the wild-derived strains carrying the Oas1b$^{Flv\text{-}r}$ allele) partial (Oas1b$^{Flv\text{-}mr}$) and full (Oas1b$^{Flv\text{-}s}$) susceptibility to flaviviral infections including Yellow Fever, Murray Valley Encephalitis virus, Dengue Fever, West Nile Fever, Japanese Encephalitis, and St. Louis Encephalitis.[121-123] Other: N/A.	susceptibility to viral diseases including flaviviruses infections
Ostm1	osteopetrosis associated transmembrane protein 1	10	melanocyte development; melanosome maturation; osteoclasts development; osteoclast ruffled border function; lysosome biology.	*Ostm1*^{gl}	grey-lethal	Intragenic deletion; 5' region of the gene including promoter, first exon and part of first intron; evidence of transposon insertion at the deletion breakpoint; LOF; recessive inheritance.	STOCK Tyr$^{c\text{-}e}$	Immune: immunodeficiency likely resulting from a stress response; thymus atrophy and lymphocyte depletion.[9] Other: early lethality (approx. 20 to 40 days of age); failure to thrive; coat color defect; osteopetrosis resulting in abnormal skeletal development; unerupted incisors; absence of marrow cavities in long bones; decreased circulating leukocyte counts; poorly regenerative anemia; dysfunctional osteoclasts (impaired acidification of Howship's lacuna); parafollicular C cells hyperplasia associated with hypocalcemia, hypophosphatemia, and vitamin D metabolism; neurodegeneration associated with neuronal accumulation of autophagosomes and lysosomal storage disorder.[124-126]	autosomal recessive osteopetrosis
Pou1f1	POU domain, class 1, transcription factor 1	16	transcription factor; pituitary development; transcriptional activation of pituitary somatotropes, lactotropes, and thyrotropes.	*Pou1f1*^{dw} *Pou1f1*^{dwJ}	*Pou1f1*^{dw}: Snell dwarf *Pou1f1*^{dwJ}: dwarf-J	*Pou1f1*^{dw}: single point mutation; G-to-T transversion; codon 261; tryptophan to cysteine conversion in the homeodomain; LOF; semidominant inheritance. *Pou1f1*^{dwJ}: chromosomal inversion or insertion of greater than 4kb in the gene; LOF; semidominant inheritance.	*Pou1f1*^{dw}: DW/J *Pou1f1*^{dwJ}: C3H/HeJ	Immune: delayed immune system maturation and development of immunocompetence; impaired T and B cell development and function; hypoplasia/atrophy of primary and secondary lymphoid organs (mainly affecting the T cell domains); partial rescue of immune defects via hormonal therapy (GH, IGF-I, prolactin, thyroxin).[127-134] Other: growth retardation; adenohypophysis hypoplasia; longevity and delayed aging resulting from somatotropic (GH/IGF-I) axis deficiency, lactotropes and thyrotropes deficiency; enhanced resistance to oxidative stress; reduced frequency of spontaneous tumor development; deafness with abnormal development and degeneration of Corti's organ; abnormal cerebellar and hippocampal development.[135-143]	panhypopituitary dwarfism; combined pituitary hormone deficiency 2
Prkdc	protein kinase, DNA activated, catalytic polypeptide	16	catalytic subunit of the DNA-dependent protein kinase; sensor for DNA damage; DNA NHEJ; DSB repair; V(D)J recombination.	*Prkdc*^{scid} *Prkdc*^{dxnph}	*Prkdc*^{scid}: severe combined immunodeficiency *Prkdc*^{dxnph}: BALB/c	*Prkdc*^{scid}: single point mutation; T-to-A transversion; premature stop codon; LOF; recessive inheritance. *Prkdc*^{dxnph}: single point mutations; A-to-G transition; methonine to valine substitution at codon 3844; C-to-T transition; arginine to cysteine substitution at codon 2140; hypomorphic; recessive inheritance.	*Prkdc*^{scid}: C.B-17 *Prkdc*^{dxnph}: BALB/c	Immune (only *Prkdc*^{scid}): V(D)J recombination defect; arrested B and T lymphocyte development at the pro-B and the pro-T cell stages; lack of mature and functional B cell and T cell; inability to mount an adaptive immune response; increased NK cell and hemolytic complement activity; leaky phenotype with generation of functional B and T cells clones (especially in old mice exposed to "dirty" environment); increased susceptibility to opportunistic bacterial infections.[144-149] Other: dysfunctional DSB repair mechanism; high frequency of thymic lymphoma development and increased susceptibility to ionizing radiation-induced tumors.[150-153]	severe combined immunodeficiency, autosomal recessive, T cell-negative, B cell-negative, NK cell-positive
Prop1	paired like homeodomain factor 1	11	transcription factor; pituitary development; transcriptional activation of pituitary	*Prop1*^{df}	Ames dwarf	single point mutation, T-to-C transition; serine to proline substitution within the α1 helix of the	Goodale large mice x pink-eyed stock, NFR/N	Immune: T cell depletion from both primary and secondary lymphoid organs; reduction in DP T cells in the thymus; abnormal presence of DP T cells in the lymph nodes; decrease in pre-, pro-, and total B cell numbers; overall impaired cell-	panhypopituitary dwarfism, combined pituitary hormone deficiency 2

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
			gonadotropes, somatotropes, lactotropes and thyrotropes.			homeodomain (amino acid 83); LOF; recessive inheritance.		mediated and humoral immunity; reduced NK activity; adiponectin-dependent reduced level of pro-inflammatory cytokines; anti-inflammatory immune skewing; partial rescue of immune defects via hormonal therapy (GH, IGF-I, prolactin, thyroxin).[130,133,134,154-156] Other: growth retardation; longevity and delayed aging resulting from somatotropic (GH/IGF-I) axis deficiency; lactotropes and thyrotropes deficiency; enhanced resistance to oxidative stress; reduced frequency of spontaneous tumor development.[133,157-159]	
Ptpn6	protein tyrosine phosphatase, non-receptor type 6	6	protein tyrosine phosphatase (PTP) family; dephosphorylation of diverse phospho-proteins for the regulation of different signaling pathways involved in hematopoiesis and immune-inflammatory cell activation; cell growth; proliferation; differentiation; regulation of glucose homeostasis.	*Ptpn6*^{me} *Ptpn6*$^{me\text{-}v}$	*Ptpn6*^{me}: motheaten *Ptpn6*$^{me\text{-}v}$: viable motheaten	*Ptpn6*^{me}: intragenic deletion; single bp; cryptic splicing site; aberrant splicing; deletion of a 101 bp segment in the encoded transcript; frameshift in the encoded protein; LOF; recessive inheritance. *Ptpn6*$^{me\text{-}v}$:single point mutation; T-to-A transversion splice consensus site; aberrant splicing; LOF; recessive inheritance.	C57BL/6J	Immune: systemic autoimmune condition; lethal pneumonitis (eosinophilic crystalline pneumonia); progressive neutrophilic dermatitis with patchy hair loss (hence the term "motheaten"); unregulated expansion of activated granulocytes and Mφs; abnormal expansion of reactive plasma cells; hypergammaglobulinemia; production of autoantibodies including anti-nuclear antibodies; systemic immune complex disease with glomerular, splenic, and skin involvement; eosinophil and mast cell hyper-responsiveness; immunodeficiency; early thymic cortical atrophy; splenic white pulp loss associated with massive EMH; loss of lymphoid follicles in lymph node and Peyer's patches; defective B and T cell maturation, activation, and expansion; overall reduced B cell population; expanded population of CD5+ B1a B cells; progressive T cell depletion; TH2-biased activation of the immune response; defective NK differentiation and function.[9,160-162] Other: failure to thrive; mean survival time of approx. 4 and 9 weeks for motheaten and motheaten viable, respectively; increased glucose tolerance and insulin sensitivity; depletion of Leydig's cells, lowered testosterone levels, and impaired spermatogenesis; decreased bone density due to unregulated osteoclast activity.[9,68,161,163-165]	N/A
Rab27a	RAB27A, member RAS oncogene family	9	Ras superfamily of GTPases; member of the Rab family of proteins; secretory granule maturation and function; regulation of vesicular fusion, trafficking and exocytotic pathway.	*Rab27a*^{ash}	ashen	single point mutation; A-to-T transversion; splice donor site downstream of exon 4; activation of two cryptic splice donor sites and the addition of an intron; LOF; recessive inheritance.	C3H/HeSn	Immune: defective secretory granule exocytosis in leukocytes including cytotoxic T cells, NK cells, eosinophils, neutrophils, and mast cells; hemophagocytic lymphohistiocytosis-like syndrome upon LCMV infection.[166-170] Other: oculocutaneous hypopigmentation; defective trafficking of melanosomes; increased bleeding times; reduction in the number of platelet-dense granules; glucose intolerance without signs of peripheral insulin resistance or insulin deficiency in the pancreas; defective exocytosis of insulin granules in pancreatic β cells; impaired release of zymogenic granules from the pancreatic acini.[166,171-174]	Griscelli syndrome type 2; hemophagocytic lymphohistiocytosis; Hermansky-Pudlak syndrome; platelet storage pool deficiency
Sharpin	SHANK-associated RH domain interacting protein	15	component of the LUBAC; substrates polyubiquitination; regulation of NF-κB signaling and TNF-induced cell death.	*Sharpin*^{cpdm} *Sharpin*$^{cpdm\text{-}Dem}$	*Sharpin*^{cpdm}: chronic proliferative dermatitis *Sharpin*$^{cpdm\text{-}}$^{Dem}: chronic proliferative dermatitis, Peter Demant	*Sharpin*^{cpdm}: intragenic deletion; single bp in the 3' end of exon 1; frameshift with premature stop codon; LOF; recessive inheritance. *Sharpin*$^{cpdm\text{-}Dem}$: single point mutation and intragenic deletion; C-to-A transition; near the 5' end of exon 1; 14 bp deletion of nucleotides 438-451; frameshift with premature	*Sharpin*^{cpdm}: C57BL/KaLawRij *Sharpin*$^{cpdm\text{-}Dem}$: OcB3/Dem	Immune: chronic progressive proliferative and eosinophilic dermatitis; clinically apparent at 3-4 weeks of age; systemic eosinophilic inflammation; eosinophilia; defects in secondary lymphoid organ development; absent Peyer's patches; spleen and lymph nodes without B cell follicles, follicular DCs and germinal centers; poorly defined B and T cell domains in the spleen with absence of marginal zone; IgG, IgA and IgE;[175,176] Other: N/A.	atopic dermatitis; hypereosinophilic syndromes

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
						stop codon; LOF; recessive inheritance.			
Slc11a1	solute carrier family 11 (proton-coupled divalent metal ion transporters), member 1	1	Mφ/monocyte-specific divalent transition metal transmembrane transporter; phagosome/phagolysosome maturation; regulation of intracellular iron metabolism; activation of Mφs, DCs, and γδ T cells; resistance to intracellular/phagocytized pathogens including *Mycobacterium* spp, *Salmonella typhimurium*, and *Leishmania donovani.*	*Slc11a1^{s};* *Slc11a1^{r}*	*Slc11a1^{s}* : pathogen susceptibility *Slc11a1^{r}*: host resistance	*Slc11a1^{s}*: single point mutation; non-conservative replacement of glycine by aspartic acid at amino acid position 169 (Gly169Asp); nonfunctional misfolded protein; LOF; recessive inheritance.	*Slc11a1^{s}:* BALB/cJ, C57BL/6J, C57BL/10J, CE/J, DBA/1J, MOLF/Ei, and NZW/LacJ *Slc11a1^{r}:* 129X1/Sv, C3H/HeN	Immune: (susceptible strains compared to resistant ones) decreased iron sequestration from phagocytized bacteria; impaired phagosome/phagolysosome maturation; impaired activation of Mφs, DCs, and γδ T cells; deficient TH1-biased immune response; reduced antigen presentation ability of bacteria-derived antigens; susceptibility to infection by several pathogens, including diverse *Mycobacterium* species, *Salmonella typhimurium*, and *Leishmania donovani*;[177-180] Other: N/A.	susceptibility to infectious diseases such as tuberculosis and leprosy; susceptibility to immunoinflammatory conditions such as rheumatoid arthritis and Crohn's disease.
Tcirg1	T cell, immune regulator 1, ATPase, H+ transporting, lysosomal V0 protein A3	19	vacuolar H^{+}-ATPase (V-ATPase), proton pump subunit; V-ATPase dependent organelle acidification; protein sorting; zymogen activation; receptor-mediated endocytosis; acidification-dependent bone resorption by osteoclasts.	*Tcirg1^{oc}*	osteosclerotic	intragenic deletion; 1579 bp region spanning exon 1 to 3; LOF; recessive inheritance.	C57BL/6J-*Vps33a^{bf}*	Immune: perturbed bone marrow microenvironment with defective hematopoiesis; increased bone marrow myelomonocytic differentiation; impaired B lymphopoiesis; abnormal expansion of B220+ and CD11b+ hematopoietic progenitors; reduced T cell activation; decreased circulating leukocyte counts.[9,181,182] Other: failure to thrive; early lethality (approx. 30 to 40 days of age); dysfunctional osteoclasts (impaired acidification of Howship's lacunae); osteopetrosis resulting in abnormal skeletal development; craniofacial deformities (shortened snout and domed skull); unerupted incisors; clubbed feet; vestibular abnormalities; absence of marrow cavities in long bones; osteopetrosis combined with rickets-like growth plate changes with failure of endochondral ossification (osteopetrorickets); parafollicular C cells hyperplasia associated with hypocalcemia, hypophosphatemia and impaired vitamin D metabolism; gastric hypochlorhydria.[9,183,184]	autosomal recessive osteopetrosis 1; clubfoot.
Tlr4	toll-like receptor 4	4	pattern recognition receptor (PRR) family; PAMPs/DAMPs recognition; activation of innate immunity; lipopolysaccharide (LPS); gram-negative bacteria; NF-κB signaling; synthesis of pro-inflammatory cytokines and chemokines; LPS-independent inflammatory responses; free fatty acids and oxidized LDL.	*Tlr4^{Lps-d}* *Tlr4$^{lps-del}$*	*Tlr4^{Lps-d}* : defective lipopolysaccharide response. *Tlr4$^{lps-del}$* : defective lipopolysaccharide response, deletion.	*Tlr4^{Lps-d}*: single point mutation; C-to-A transversion; histidine to proline substitution at position 712; LOF; codominant inheritance. *Tlr4$^{lps-del}$*: intragenic deletion, 7472 3bp region encompassing the entire *Tlr4* locus; LOF; recessive inheritance.	*Tlr4^{Lps-d}*: C3H/HeJ *Tlr4$^{lps-del}$*: C57BL/10ScN	Immune: impaired recognition of TLR4-specific PAMPs and DAMPs including LPS, free fatty acids, and HSP60; defective activation of the innate immunity against specific bacterial, fungal and viral infections; abnormal regulation of TH1 vs TH2 immune response by DCs in response to TLR4-specific PAMPs and DAMPs;[9,185-191] protection from experimentally-induced autoimmune and immunoinflammatory conditions including GVHD, EAE, CIA, DSS colitis, and endotoxic shock.[192-196] Other: protection from experimentally-induced alcoholic steatohepatitis, atherosclerosis, diabetes, insulin resistance, ischemia-reperfusion injury, metabolic syndrome, myocardial infarction, obesity, renal fibrosis, stroke, and traumatic injury;[197-205] delayed skin wound healing;[206] increased susceptibility to cutaneous chemical carcinogenesis.[207]	sepsis; cytokine storm; gram negative bacteria infections.
Tnf	tumor necrosis factor	17	multifunctional proinflammatory cytokine; TNF superfamily; innate immune response; chronic inflammation; endogenous pyrogen; apoptotic cell death; cachexia; acute phase response.	*Tnf^{Bpsm1}*	bone phenotype spontaneous mutation 1	retrotransposone insertion into the 3' UTR; polyadenylation signal with premature termination of the transcript; GOF; dominant inheritance.	unspecified; it involves BALB/c, C57BL/6 and 129/Sv	Immune: deregulated expression of mutant *Tnf* by myeloid derived immune/inflammatory cells; increased TNF signaling in synoviocytes; progressive polyarthritis mainly affecting the interphalangeal, metacarpophalangeal/metatarsophalangeal, carpo-metacarpal/tarso-metatarsal, and carpal/tarsal joints; absence of IgM or IgG rheumatoid factors; progressive inflammation, thickening, and fibrosis of the aortic and mitral valves; aortic root aneurysms (only observed on the BALB/c background).[208]	rheumatoid arthritis

Gene/Locus symbol	Gene/Locus name	Chr	Function	Mutation or allelic variant symbol	Mutation or allelic variant name	Mutation or allelic variant detail	Original/affected strain/stock	Phenotype	Human disease models
								Other: N/A.	
unknown	unknown	1	unknown	*Dh*	dominant hemimelia	unknown mutation; semidominant inheritance.	crossbred stock	Immune: both homozygous and heterozygous mutants affected by congenital asplenia; heterozygous characterized by enlarged lymph nodes with decreased number of B cells and massive expansion of T cells; impaired T cells maturation; leukocytosis; thrombocytosis; decreased serum levels of IgM and IgG2.[209] Other: early postnatal lethality in homozygous; defect in splanchnic mesoderm development; gastrointestinal and urogenital anomalies; skeletal abnormalities; hind limb malformations (hence hemimelia); reduction in the vertebral number.[9,210,211]	congenital asplenia
unknown	unknown	Y	unknown	*Yaa*	accelerated autoimmunity and lymphoproliferation transposition; accelerated autoimmunity and lymphoproliferation	insertion mutation; 4 Mbp telomeric segment of the chrX translocated into the pseudoautosomal region of chrY; duplication about 19 genes including *Tlr7*; GOF; Y-linked inheritance.	BXSB/MpJ	Immune: promoting and/or accelerating autoimmune phenotypes in autoimmune-prone backgrounds (e.g. MRL/MpJ and NZW/LacJ) and mouse models including MRL/MpJ-Fas^{lpr}; translocation of *Tlr7* from the chrX into the chrY; duplication and upregulation/overexpression of *Tlr7* (*Xist* normally inactivates one copy of *Tlr7* in females); increased TLR7 signaling in response to single-stranded RNA molecules; accelerated onset of autoimmunity in BXSB/MpJ males (50% mortality at 6 months) when compared to females (50% mortality at 20 months); systemic lupus erythematosus-like condition; CD4 T cell expansion and activation; overexpression of IL21; unregulated T_{FH} activation resulting from defective CD8+ Treg cell function; generalized lymphoid hyperplasia (involving both LNs and spleen); overall thymic cortical depletion with reduced epithelial network associated and lower number of thymocytes; monocytosis; hypergammaglobulinemia; hemolytic anemia; production of autoantibodies including anti-nuclear and anti-erythrocyte antibodies; severe immune complex-mediated GN and vasculitis.[9,212-218] Other: N/A.	systemic lupus erythematosus
Zap70	zeta-chain (TCR) associated protein kinase	1	member of the protein tyrosine kinase family; part of the T cell receptor; development of T lymphocytes and thymocytes; T lymphocyte receptor-mediated signal transduction.	*Zap70st*	strange	single point mutation, C-to-T transition; arginine to cysteine substitution at codon 464; disruption of a highly conserved motif within the kinase domain; LOF; recessive inheritance.	B6.129S2-*Cd28^{tm1Mak}*/J	Immune: thymus hypoplasia; absence of mature T cells; thymocyte development is arrested at the $CD4^+$ $CD8^+$ DP stage of differentiation; impaired TCR signaling.[219] Other: N/A.	N/A
Zap70	zeta-chain (TCR) associated protein kinase	1	member of the protein tyrosine kinase family; part of the T cell receptor; development of T lymphocytes and thymocytes; T lymphocyte receptor-mediated signal transduction.	*Zap70^{m1Saka}*	Shimon Sakaguchi	single point mutation; G-to-T transversion at nucleotide 489; tryptophan to cysteine substitution at codon 163; hypomorphic; LOF; recessive inheritance.	BALB/cJ	Immune: altered thymic T cell selection; T cell–mediated autoimmune polyarthritis mainly affecting the interphalangeal, metacarpophalangeal/metatarsophalangeal, carpo-metacarpal/tarso-metatarsal, and carpal/tarsal joints; interstitial pneumonitis; systemic vasculitis; granulomatous panniculitis; high titers of rheumatoid factor; autoantibodies specific for type II collagen; severe hypergammaglobulinaemia; high concentration of circulating immune complexes; induction of severe polyarthritis, interstitial pneumonitis, and ileitis after challenge with polysaccharides from *Saccharomyces cerevisiae*.[220-222] Other: N/A.	rheumatoid arthritis

Abbreviations and acronyms used in the table:

A: adenine, AICD: activation-induced cell death, BCR: B cell receptor, bp: base pair, C: cytosine, Chr: chromosome, CIA: collagen-induced arthritis, CNS: central nervous system, CTL: cytotoxic T lymphocytes, DAMPs: damage-associated molecular patterns, DC: dendritic cell, DN: double negative, DSB: double strand break, DSS: dextrane sodium sulfate, EAE: experimental autoimmune encephalitis, EMH: extramedullary hematopoiesis, FSH: follicle stimulating hormone, G: guanine, GC: germinal center, GH: growth hormone, GN: glomerulonephritis, GOF: gain of function, GVHD: graft versus host disease, IFN: interferon, Ig: immunoglobulin, IGF-I: insulin-like growth factor 1, LCMV: lymphocytic choriomeningitis virus, LH: luteinizing hormone, LOF: Loss of function, LPS: lipopolysaccharide, Mφ: macrophage, LUBAC: linear ubiquitin assembly complex, N/A: not applicable, not available, NF-κB: nuclear factor kappa-light-chain-enhancer of activated B cells, NHEJ: non-homologous end joining, NK: natural killer, PAMPs: pathogen-associated molecular patterns, RPE: retinal pigmented epithelium, ssDNA: single strand DNA, T: thymine, TAM: tumor associated macrophage, TCR: T cell receptor, TGF: transforming growth factor, TI: T independent antigen, TLR: Toll-like receptor, TNF: tumor necrosis factor.

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